ARCHIVES OF PEDIATRICS

January 1939

Published every month since 1884

Volume 76 Number 1

even when the causative organism may be a "persistent staph"

Cosa-Signemycin*

GLUCOSAMINE POTENTIATED TETRACYCLINE WITH TRIACETYLOLEANDOMYCIN

increases the certainty of safe, rapid response

AS PROVED BY extensive clinical trials—an over-all success rate of more than 94% was achieved in a total of 3,280 cases.†

AS PROVED BY effectiveness in "problem infections"—a response rate better than 96% was recorded in a group of 221 gastrointestinal infections including chronic intestinal amebiasis; 91% of 465 urogenital infections were successfully controlled.†

AS PROVED BY success in mixed infections—more than 95% of 1,000 acute and chronic respiratory tract infections were successfully treated; a 99% cure rate was achieved in mixed bacterial pneumonias.†

AS PROVED BY excellent safety record—extremely well tolerated; discontinuance of medication was necessary in only 11 of 3,820 patients.†

A significant number of the above cases had not responded to other antibiotics.

Cosa-Signemycin is particularly valuable in home and office, where susceptibility testing is difficult or impractical.

SUPPLY: *Capsules* (green and white), 250 mg. and 125 mg.

New Oral Suspension (raspberry-flavored) 2 oz. bottle, 125 mg. per teaspoonful (5cc.)

New Pediatric Drops (raspberry-flavored) 10 cc. bottle, 5 mg. per drop, plastic calibrated dropper.

Average dosage: For adults, 1-2 Gm. daily in divided doses; proportionately less for children, depending on age, weight, and severity of infection.

†Literature and bibliography available on request.

*Trademark

Pfizer *Science for the world's well-being*

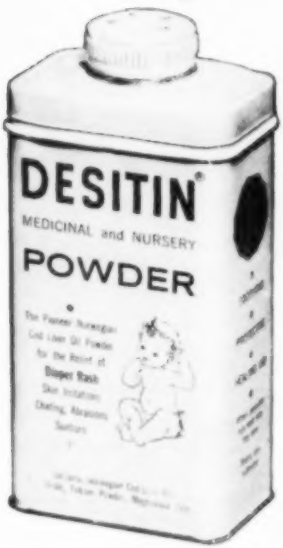
PFIZER LABORATORIES,
Division, Chas. Pfizer & Co., Inc.
Brooklyn 6, N. Y.

ONLY

DESITIN®

Medicinal and Nursery

POWDER



is saturated with high grade Norwegian Cod Liver Oil (plus its vitamins A and D)... so will not "dry out" the skin, nor deprive it of its natural fats.

Instead... DESITIN POWDER acts to keep baby's skin normally lubricated, soft, smooth, **supple** ..... in **diaper rash**

prickly heat rash
chafing, skin irritations

Every baby deserves the tender, loving care of soothing, protective, **healing** DESITIN POWDER.

Mothers will appreciate your recommendation of both DESITIN POWDER and DESITIN OINTMENT.

samples cheerfully sent—write **DESITIN CHEMICAL COMPANY**
812 Branch Ave., Providence 4, R. I.

DESITIN SOAP ... ideal for baby's bath!

ARCHIVES OF PEDIATRICS

Vol. 76 No. 1

January 1959

ALBRO C. GAYLOR, *Publisher*

JOHN FITCH LANDON, *M.D., Editor*

Editorial Board

Philip M Stimson, M.D., *New York*
Frederick H. Wilke, M.D., *New York*
P. W. Braestrup, M.D., *Denmark*
Ronald N. Maclean, M.D., *Argentina*
John Zahorsky, M.D., *St. Louis*
J. Frederick Eagle, Jr., M.D., *New York*
Y. Nishizawa, M.D., *Japan*
Roald Rinvik, M.D., *Norway*

Associate Editors

Michael A. Brescia, M.D., *New York*
Catherine E. Spears, M.D., *New Jersey*
Herberr V. von Gal, M.D., *Connecticut*

CONTENTS

Coxa Plana
Beckett Howorth, M. D. 1

Pediatric Progress During the Last Twenty-five Years
Murray H. Bass, M.D. 17

Clinical Conference: Congenital Cerebral Aneurysms and Coarctation of the Aorta
The Mt. Sinai Hospital, New York 28

Current Literature 44

Cover — Abraham Jacobi (1830-1919) — Page 8
(Coyright "Pioneers of Pediatrics")

*Copyright, 1959, by E. B. Treat & Co., Inc., 45 East 17th St., N. Y. 3.
All Rights Reserved. Entered as second class matter Feb. 5, 1892,
at New York, N. Y., P. O. under the Act of March 3, 1879.*

Published monthly since 1884.
Subscriptions: one year—$7.50; two years—$12.00; three years—$16.00.

[Reproduction of cover of first issue]

Vol. I. JANUARY 15, 1884. No. 1.

THE

Archives of Pediatrics

A MONTHLY JOURNAL DEVOTED TO THE

DISEASES OF INFANTS AND CHILDREN.

EDITED BY

WILLIAM PERRY WATSON, A. M., M. D.,

Physician for Diseases of Children to the Central Dispensary and Assistant to Christ Hospital, Jersey City, N. J.

COLLABORATORS:

J. LEWIS SMITH, M. D.,
NEW YORK.

J. M. KEATING, M. D.,
PHILADELPHIA.

JOS. P. OLIVER, M. D.,
BOSTON.

F. FORCHHEIMER, M. D.,
CINCINNATI.

EDW. BORCK, M. D.,
ST. LOUIS.

MARCUS P. HATFIELD, M. D.,
CHICAGO.

WM. T. PLANT, M. D.,
SYRACUSE.

HENRY D. INGRAHAM, M. D.,
BUFFALO.

JEROME WALKER, M. D.,
BROOKLYN.

JAMES F. GOODHART, M. D.,
LONDON.

HENRY ASHBY, M. D.,
MANCHESTER.

JAMES FINLAYSON, M. D.,
GLASGOW.

A. D. BLACKADER, M. D.,
MONTREAL.

THREE DOLLARS PER ANNUM, IN ADVANCE.

Entered at the Post Office, Jersey City, N. J., as second-class matter.

NEW CLINICAL EVIDENCE

offers additional proof that

CREAM of RICE

IS EASIER TO DIGEST

than any other kind of cereal!

In a study conducted with intubated men and women, each received a cup of Cream of Rice, oatmeal and farina on different days. From each subject samples were withdrawn periodically and tested for sugar. The amount of sugar tested was taken as the index of digestibility.

Results showed that Cream of Rice was easier to digest and gave quicker food energy than the other cereals tested.

RECOMMENDED BY LEADING PEDIATRICIANS

More and more pediatricians are recommending Cream of Rice as one of baby's first solid foods. Pediatricians are also recommending it for growing children—because it's so rich in food value.

Both adults and children love the deliciously different taste of Cream of Rice. That's why it's so good as a breakfast cereal, too!

Cream of Rice is non-allergenic, low in sodium, low in fat—rich in Vitamin B_1, Riboflavin, Niacin and Iron.

Cooks in ½ minute!

WRITE FOR PROFESSIONAL SAMPLES TO:
GROCERY STORE PRODUCTS CO.
Dept. CIA, West Chester, Pa.

modern once-a-day sulfa therapy in

NEW CHERRY-FLAVORED KYNEX* ACETYL

N¹Acetyl Sulfamethoxypyridazine Lederle

PEDIATRIC SUSPENSION...

In a wide range of infections caused by sulfa-sensitive organisms, delicious new KYNEX Suspension is fully effective. Therapeutic blood levels are rapidly reached and sustained on one easy-to-take dose a day.

Young patients with urinary tract infections, bacillary dysenteries and many other common disorders, respond readily, improve steadily and before long are back on their feet.

DOSAGE IN CHILDREN: Recommended first day dosage is 1 teaspoonful (250 mg.) for each 20 lb. body weight administered immediately after a meal. The dosage for each day thereafter is ½ teaspoonful (125 mg.) for each 20 lbs. administered once a day following a meal.

SUPPLIED: Bottles of 4 fl. oz. Each teaspoonful (5 cc.) of cherry-flavored suspension contains 250 mg. of sulfamethoxypyridazine activity.

LEDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

Reg. U. S. Pat. Off.

Editorial and Business Communications . . .

E. B. Treat & Co., Inc.,
45 East 17th St., New York 3, N. Y.

Subscription:
$7.50 a Year
Foreign: $8.25
Single Copy: $1.00

Original Articles—brief reports of rare and interesting cases or new modes of treatment are solicited. None will be considered for publication except with the distinct understanding that it is contributed solely to this journal. Manuscripts must be typewritten and double-spaced. Please submit only the original copy. The publishers will not be responsible for author's views.

Illustrations—those deemed necessary by the publishers will be furnished free when clear and distinct photographs are supplied. Drawings must be in India ink on white paper.

Copyright—this journal's copyright protects original material; therefore none can be reproduced without publishers' permission.

Reprints—prompt orders for reprints of original articles will be accepted. Schedule of prices will be sent upon request to publishers.

Discontinuances—written notice of discontinuance of subscriptions must be addressed to publishers.

Remittances—payment by check, draft, post office or express money order —please.

Change of address—please give six weeks' notice, with precise name under which subscription is entered, including both old and new addresses and current zone number.

The Cover-Abraham Jacobi (1830-1919)

It seems significant that this *75th Anniversary* issue of *Archives of Pediatrics* should present on its cover, a portrait of the "Father of American Pediatrics". The long list of Dr. Jacobi's pioneering distinctions, singular pediatric achievements, the full complement of academic and professional honors bestowed upon him . . . all are too well known for recording here.
Archives recalls with pride that for more than twenty-five years, Dr. Jacobi was numbered among its illustrious editors.

your patients won't resist it most common Gram-positive infections can't

In a recent evaluation of TAO in pediatric patients, 85% (34 of 40 patients) were afebrile within 48 hours and their infections cleared.[1]

"NO ANTIBIOTIC TASTE"

TAO is "practically tasteless."[2] Adding delicious cherry flavor to the TAO formula makes it widely acceptable to children.

"NO ADVERSE HEMATOLOGICAL REACTIONS ON [TAO]"[1]

"No serious toxicity developed in any of the 40 patients studied... even after using TAO for 31 days. Mild side effects in 3 instances (1 rash, 2 vomiting) were fully controlled when therapy was discontinued."[1]

DOSAGE AND ADMINISTRATION: Dosage varies according to the severity of the infection. *For adults,* the average dose is 250 mg. q.i.d.; to 500 mg. in more severe infections. *For children* 8 months to 8 years of age, a daily dose of approximately 30 mg./Kg. body weight in divided doses has been found effective. Since TAO is therapeutically stable in gastric acid, it may be administered without regard to meals.

SUPPLIED: TAO Capsules—250 mg. and 125 mg., bottles of 60. TAO for Oral Suspension—1.5 Gm.; 125 mg. per teaspoonful (5 cc.) when reconstituted; unusually palatable cherry flavor; 2 oz. bottle.

References: 1. Mellman, W. J., et al.: Paper presented at Symposium on Antibiotics, Washington, D. C., October 15-17, 1958. 2. Wennersten, J. R.: Antibiotic Med. & Clin. Therapy 5:527 (Aug.) 1958.

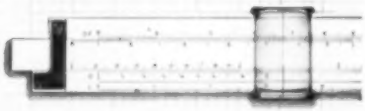
PRONOUNCED TAY-O

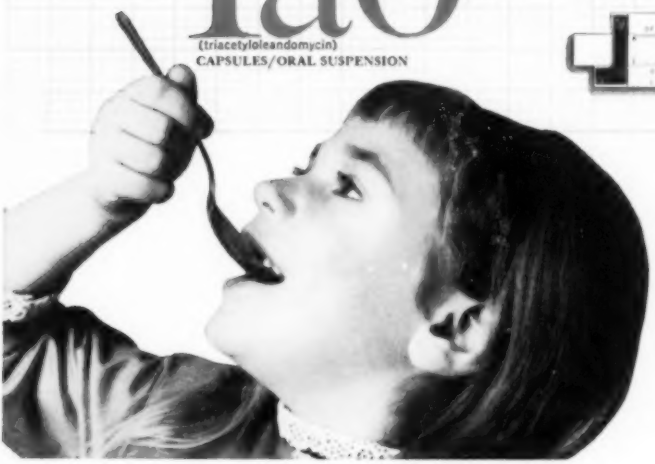
Tao*

(triacetyloleandomycin)

CAPSULES/ORAL SUSPENSION

Designed for superior control of common Gram-positive infections





PFIZER Rx

Science for the World's Well-Being

Division, Chas. Pfizer & Co., Inc.

New York 17, N. Y.

"Much better-

the COSAsaur is the family emblem of the COSA antibiotics. It symbolizes the natural origin of glucosamine, a substance older than man himself. Glucosamine is widely distributed throughout nature—in plants and seashells, in body tissues and mother's milk. Today, as in the dinosaur era, "Cosa" is basic to life.

Proven in research

1. Highest tetracycline serum levels
2. Most consistently elevated serum levels
3. Safe, physiologic potentiation (with a natural human metabolite)

And now in practice

4. More rapid clinical response
5. Unexcelled toleration

REFERENCES: 1. Carlozzi, M.: Antibiotic Med. & Clin. Therapy 5:146 (Feb.) 1958. 2. Welch, H.; Wright, W.W., and Staffa, A.W.: Antibiotic Med. & Clin. Therapy 5:52 (Jan.) 1958. 3. Marlow, A.A., and Bartlett, G.R.: Glucosamine and leukemia, Proc. Soc. Exp. Biol. & Med. 84:41, 1953. 4. Shalowitz, M.: Clin. Rev. 1:25 (April) 1958. 5. Nathan, L.A.: Arch. Pediat. 75:251 (June) 1958. 6. Cornbleet, T.; Chesrow, E., and Barsky, S.: Antibiotic Med. & Clin. Therapy 5:328 (May) 1958. 7. Stone, M.L.; Sedlis, A., Bamford, J., and Bradley, W.: Antibiotic Med. & Clin. Therapy 5:322 (May) 1958. 8. Harris, H.: Clin. Rev. 1:15 (July) 1958.

-thank you, doctor"

COSA-TETRACYN*

GLUCOSAMINE-POTENTIATED TETRACYCLINE

CAPSULES (black and white) 250 mg., 125 mg. (for pediatric or long-term therapy)

ORAL SUSPENSION (orange-flavored) 125 mg. per tsp. (5 cc.), 2 oz. bottle

NEW! PEDIATRIC DROPS (orange-flavored) 5 mg. per drop, calibrated dropper, 10 cc. bottle

COSA-TETRASTATIN*

glucosamine-potentiated tetracycline with nystatin

Antibacterial effectiveness plus added protection against monilial superinfection

CAPSULES (black and pink) 250 mg. Cosa-Tetracyn *plus* 250,000 u. nystatin

ORAL SUSPENSION 125 mg. per tsp. (5 cc.) Cosa-Tetracyn, *plus* 125,000 u. nystatin, 2 oz. bottle

COSA-TETRACYDIN*

glucosamine-potentiated tetracycline-analgesic-antihistamine compound

For relief of symptoms and malaise of the common cold and prevention of secondary complications

CAPSULES (black and orange) – each capsule contains: Cosa-Tetracyn 125 mg.; phenacetin 120 mg.; caffeine 30 mg.; salicylamide 150 mg.; buclizine HCl 15 mg.

Pfizer *Science for the world's well-being*

PFIZER LABORATORIES

Division, Chas. Pfizer & Co., Inc., Brooklyn 6, New York

*Trademark

Memo from the publisher . . .

A brief message in the September 1958 issue told something of the constructive developments during the nine months under my ownership and management. It underscored appreciation of the personal efforts made by *Archives'* editors, acknowledged the increasingly valuable contributions, and thanked the growing of doctor-subscribers whose active readership support continues to be registered.

Now in this 75th Anniversary number, it seems fitting to record not just what we have done—but to reaffirm that we will go on building up to steadily higher levels . . . that we mean to make the "new" *Archives* into the kind of practical journal we realize is much needed in the pediatric and allied-interests' field.

Albro C. Gaylor, Publisher

PROTECT Little Braves' Bottoms

WITH Diaparene® CREME Peri-Anal® SPECIFIC For Stool Irritations

"DIAPARENE PERI-ANAL is an efficient and safe agent in the prevention and treatment of perianal dermatitis"* . . . newborn "sore-bottom" due to loose, transitional stools and irritations caused by diarrhea or loose stools following oral antibiotic therapy.

*Grossman, Leo, "*A New Specific Treatment for Perianal Dermatitis*", Arch. Ped., 71:173-79, June, 1955*

ANOTHER FINE PEDIATRIC SPECIALTY BY BREON

U. S. PAT. NO. 2,843,522

HOMEMAKERS PRODUCTS DIVISION, George A. Breon & Company, 1450 Broadway, New York 18, N.Y.

stop diarrhea

solves acute diarrheal disease problems...

■ swiftly relieves symptoms ■ rapidly destroys bacterial pathogens (bactericidal rather than bacteriostatic) ■ succeeds where others fail against the enteric "problem pathogens" — increasingly prevalent, refractory strains of Staphylococcus, Escherichia, Salmonella and Shigella

...without creating new problems

■ does *not* upset the balance of normal intestinal flora
■ does *not* encourage monilial or staphylococcal overgrowth
■ does *not* induce significant bacterial resistance

Furoxone® liquid

BRAND OF FURAZOLIDONE

A PLEASANT ORANGE-MINT FLAVORED SUSPENSION containing FUROXONE, 50 mg. per 15 cc., with kaolin and pectin ■ For patients of all ages (may be mixed with infant formulas, passes through a standard nursing nipple) ■ Dosage: Should provide (in 4 divided doses) 400 mg. daily for adults, 5 mg./Kg. daily for children ■ Supplied: bottles of 240 cc. (also: FUROXONE Tablets, 100 mg. scored, bottles of 20 and 100)

THE NITROFURANS—a unique class of antimicrobials
EATON LABORATORIES, NORWICH, NEW YORK

"Ah, to Breathe Again!"

Open spaces are no help in breathing, when nasal passages are shut tight from pollen or with head colds.

Neo-Synephrine HYDROCHLORIDE

Pediatric Nasal Spray

0.25% • 20 cc.

contains Zephiran® chloride 1:5000, antibacterial wetting agent and preservative for greater efficiency

In the handy plastic squeeze bottle

Prompt and Prolonged Decongestion

- Usually no compensatory vasodilation
- No sting
  Virtually no systemic reactions

Winthrop LABORATORIES
NEW YORK 18, N. Y.

Neo-Synephrine (brand of phenylephrine), trademark reg. U.S. Pat. Off.

COXA PLANA

BECKETT HOWORTH, M.D.*

Connecticut

Coxa Plana is a disease of the hip occurring in children between the ages of four and ten years. It is sometimes called osteochondritis deformans coxae juvenilis, Legg's, Perthes, Calve's, or Waldenstrom's disease. It was described independently in 1910 by Legg Perthes, and Calvé, who distinguished it from tuberculosis, whereas Waldenstrom in 1909 had considered it a form of mild bone tuberculosis. He later gave it the name coxa plana. This term describes the flattening of the head of the femur which occurs as the disease advances but is not adequate to describe the entity, nor the changes which occur before the flattening. However, no better name has yet been proposed.

The disease is characterized by degenerative changes in the capital epiphysis of the femur, and runs a self-limited course. While there are many individual variations, the basic features and course of the disease are characteristic and should rarely be confused with any other lesion.

Coxa plana is one of a group of diseases, the osteochondroses, which affect various epiphyses at various ages. Slipping of the Upper Femoral Epiphysis, another member of the group, occurs in children between the ages of eight and eighteen. Many of its features are similar to those of coxa plana. These two diseases of the hip are the most common and most serious of the osteochondroses, as they are likely to result in persistent pain, restrictions of motion, and disability. The osteochondrosis of the tibial tuberosity is called Osgood-Schlatter disease, that of the patella, Larsen-Johansson disease, and Blount-Barber disease of the upper tibial epiphysis appears to belong in the same group. The osteochondroses of the foot include apophysitis of the calcaneus, Kohler's disease of the tarsal navicular, and Freiberg's infraction of the second metatarsal head. Interestingly, the upper extremity is rarely affected by this type of lesion, Kienbock's disease of the carpal lunate, which occurs in adults, being the only distinct member of the group. Epiphysitis

* Chief of Orthopedics, Greenwich Hospital, Greenwich, Conn. Clinical Professor Orthopedics, N. Y. Univ. Post-Graduate Medical Center. Consultant in Orthopedics, Roosevelt Hospital, New York City. Consultant in Orthopedics, Newington Home & Hospital for Crippled Children, Newington, Conn. Consultant in Orthopedics, Northern Westchester Hospital, Mt. Kisco, N. Y.

of the spine, or Scheuermann's disease appears to belong to this group also. All the members of the group are characterized by a vascular disturbance with swelling of the soft tissues and degenerative changes in the affected bone and cartilage. While much has been learned of the clinical features of these diseases in the past four or five decades and treatment has improved, much remains to be learned as to etiology, prevention and even effective treatment. Roentgenography has been of inestimable value in the diagnosis of coxa plana and in following its course and the effects of treatment.

ETIOLOGY

Coxa plan is not uncommon in orthopedic or even in pediatric practice. In the ten years 1935-'44 inclusive, one hundred and fifteen cases were seen at the New York Orthopedic Hospital, and have been reported. Coxa plana is much more common in boys than in girls, in a ratio of about six to one. It occurs with equal frequency in either hip and about fifteen percent of the cases are bilateral. The age incidence is four to ten years, but it may be seen in younger children under treatment for congenital dislocation of the hip. It is also seen in young cretins. There are various opinions as to whether the lesion should be properly called coxa plana when it occurs with congenital dislocation of the hip or cretinism, but there seems to be no essential difference. Degenerative changes similar to those seen in coxa plana may occur in older children in the course of treatment of slipping of the upper femoral epiphysis, and in adults with fracture of the neck of the femur, also due to vascular damage.

The cause of the vascular disturbance which is responsible for coxa plana is not definitely known. The disease is rarely seen in more than one child in a family, and no definite hereditary or familial relationship has been found. Congenital abnormality of the acetabulum has been given as the cause of the disease by some, such as Murk Jansen, but the evidence does not support this conclusion. Injury to the hip or leg is often described as related to the onset of symptoms, but the injury is usually trivial, and it should be remembered that children of this age group are active and constantly having minor trauma. The lesion has been ascribed to infection, but no infection has been demonstrated in the hip, and there has been little to support the idea of secondary infection. Endocrine imbalance has been thought in certain cases to be responsible for the disease, partly because some of the children are overweight, but this is not an uncommon characteristic of children at this age, and no definite endo-

crine disturbance has been proven. Cretinoid epiphyseal dysgenesis seems to bear no direct etiologic relation to the ordinary type of coxa plana, nor does hypothyroidism in general. Changes similar to coxa plana are sometimes seen in hips affected by achondroplasia, chondryodysplasia, juvenile rickets and Morquio's disease but again there is no relation to ordinary coxa plana. The possibility of an allergic or toxic reaction, or a vitamin disturbance has been considered but no evidence has been adduced. It appears quite possible that the vascular disturbance may be initiated by any one of several different causes.

PATHOLOGY

There has been little opportunity to study the pathology of coxa plana directly, especially in the early stages of the disease. Accordingly, roentgenograms have been the sole basis for interpretation of the pathology in most reports. An ideal pathologic study would include large serial sections of the head and neck of the femur, including the soft tissues, in all stages of the disease. However, such a study would be next to impossible in humans and has not been practical in experimental animals.

Opportunity has been presented to study the pathology of the soft tissues and of drillings from the bone in connection with surgical operations in 60 cases. In the early stages of the disease synovitis and periostitis were found, with swelling, edema, and hypervascularity. Perivascular lymphocytic infiltration, and scattered plasma cells and large mononuclear wandering cells were demonstrated in miscroscopic sections. Decalcification and increased vascularity at the junction of the neck and the epiphyseal disc result in softening. Dense areas soon appear in the capital epiphysis, usually at the center, representing bone condensation. Later these dense areas are invaded by blood vessels and granulation tissue which tend to absorb them, leaving areas of rarefaction. As the internal support of the capital epiphysis is lost, the head begins to flatten and widen, or mushroom. At the same time the neck becomes thicker, while its longitudinal growth is slowed. The soft tissues in the meantime become less edematous and less vascular, scarred and inelastic, and a pannus encroaches upon the margin of the head. As the disease heals the ossification center of the head becomes recalcified and the density of the proximal end of the neck is restored. Slowly the bone becomes trabeculated, but the mushroom shape is not lost. As a result of this deformity there is incongruity of the head and aceta-

bulum, and the smooth ball and socket motion is lost. The acetabulum tends to become deformed to conform somewhat to the deformity of the head, whereas the enlarged head protrudes beyond the lateral margin of the acetabulum. Five to seven years are required for complete healing in most cases.

Some years after the disease heals the early changes of osteoarthritis are seen, and develop gradually over a period of years. Eventually the osteoarthritis may be severe, in middle or later life.

STAGES

Coxa plana may be divided into four stages: the incipient or early stage, lasting several weeks, characterized by changes in the soft tissues rather than the bones, with mild symptoms and signs due to synovitis. (2) The active or progressive stage, lasting one to two years, with degenerative changes in the head and softening at the ephiphyseal disc. (3) the healing stage, lasting three or four years in which the dense areas in the ossification center of the capital epiphysis are slowly replaced by granulation tissue which is gradually converted into normal bone while the soft tissues become sclerotic and the symptoms and signs of synovitis subside. (4) The residual stage in which only the deformity with its related symptoms remains. Osteoarthritis may develop in this stage. The soft tissue changes precede the bone changes in each period.

SYMPTOMS

Early and active stages: the symptoms in these stages are those of synovitis, limp, pain, limitation of motion and disability. Limp is a common symptom, and is due to protection of the hip. The pain is usually mild and inconstant. It is referred to the groin or inner side of the thigh or knee, following the distribution of the obturator nerve. The pain may be aggravated by vigorous activity or injury, relieved by rest. Restriction of motion may be noticed, as in tying the shoe laces or crossing the legs. Disability for sports or other vigorous activity is usually present; rarely is the pain severe enough to prevent walking.

In the healing stage the acute symptoms gradually diminish and are replaced by symptoms due to the faulty mechanics associated with the deformity. Limp is now due to shortening and restriction of motion. There may be slight pain on vigorous activity, slight stiffness and associated disability. In adult life the symptoms of osteocarthritis may supervene, with increasing limp, pain, stiffness and disability.

SIGNS

Early stage: the signs in this stage are those of synovitis: limp, limitation of motion with pain at the extremes, weakness. Limitation of motion is usually slight, but lateral motion and rotation are affected more than flexion. Pain and spasm at the extremes of motion are characteristic, varying in intensity with the acuteness of the disease. Sometimes there is tenderness anterior to the joint. The pain and spasm tend to subside with rest, and motion may improve. In time there is slight atrophy of the thigh, and as the head begins to flatten femoral shortening appears. The shortening is usually one quarter to one half inch. As the disease heals the signs of synovitis are replaced by restriction of motion, especially abduction and internal rotation, due to the incongruity of the joint. These signs become more pronounced as osteoarthritis develops.

LABORATORY TESTS

The laboratory tests are chiefly of value in differential diagnosis, for the urinalysis, blood count, blood calcium, phosphorus and phosphatase and the basal metabolic rate are normal in coxa plana. The erythrocyte sedimentation rate is usually slightly increased, averaging 25 mm. per hour, occasionally as high as 40 mm. Cultures of the synovia, synovial membrane and bone have been negative.

ROENTGENOGRAMS

The first roentgenographic evidence of the disease is a globular swelling of the capsule. A few weeks after the onset the epiphyseal line appears wider and there is irregular decalcification at the proximal end of the neck. The joint space appears wider inferiorly because of the slight lateral subluxation. It appears wider superiorly because of flattening of the ossified portion of the capital epiphysis. However there is no change in the shape of the articular cartilage at this stage. An irregular dense area soon appears in the capital epiphysis, usually in the central or lateral portion. Gradually this dense area becomes irregularly decalcified but it may become more extensive at the same time. This appearance is sometimes called "fragmentation", but this term is a misnomer and should be avoided. During the period of irregular decalcification the head becomes thinner, flatter and wider and the neck thicker and shorter. The soft tissue swelling gradually subsides, the proximal end of the neck recalcifies, becomes convex and grows into the head.

Recalcification of the decalcified portion of the head progresses more slowly. Eventually normal trabeculation occurs, but the head retains its mushroom shape and the subluxation persists. The shape of the acetabulum usually tends to conform to the shape of the head. Osteoarthritic changes may be seen in middle or later life.

DIFFERENTIAL DIAGNOSIS

Any child between the ages of three and ten years, complaining of a limp and pain in the groin, anterior or medial aspect of the thigh or inside of the knee should be suspected of having coxa plana. The hips should be examined carefully for restriction of motion, pain and spasm. Anterior or posterior and lateral roentgenograms should be made. If the diagnosis is in doubt the child should be put to bed until the physical signs disappear and later roentgenograms reveal no evidence of coxa plana. Laboratory tests may be an aid in eliminating other diseases, but the roentgenographic appearance of coxa plana in its various stages is quite characteristic.

Coxa plana occurring in the course of treatment of congenital dislocation of the hip exhibits the characteristic features of both diseases unless the hip has been completely reduced before structural changes occur. Usually the history is definite. Cretinoid epiphyseal dysgenesis is more often seen in younger infants, there are also signs of hypothyroidism, and the therapeutic response is prompt. Suppurative arthritis of the hip can be distinguished from

ROENTGENOGRAMS—FIG. 1

Fig. 1. Typical coxa plana, with early diagnosis, in a child of 7 years, treated with drilling operation. Pain had been present for two months. Other symptoms included limp, considerable limitation of motion, pain and spasm. Good healing had taken place by four years after onset—less than the average time—despite no treatment during the first fifteen months and extensive involvement.

a . . . July 16, 1930. Two months after onset, the capsule is swollen. The epiphyseal line is wide, with decalcification at the junction with the neck; the neck is wide. The head is flat superiorly; the articular surface is uneven; ossification is irregular; density is increased. The joint space is wide. No treatment was given for fifteen months.

b . . . Dec. 1, 1930. After seven months, the head is flatter, thinner, denser and more irregularly ossified. The epiphyseal line is wider, more irregular, and decalcified. Slight subluxation is shown.

c . . . Mar. 2, 1931. Ten months after onset, the head is still flatter, thinner, and more irregularly ossified. The epiphyseal line is beginning to reossify. One third of the head is outside the acetabulum.

d . . . Oct. 14, 1931. After seventeen months, the lateral portion of the head is almost absorbed. The epiphyseal line has healed. This is the end of the absorptive phase. At operation, on Oct. 22, 1931, six holes were drilled. The patient remained in bed for four months, then was ambulatory in short spica for two months.

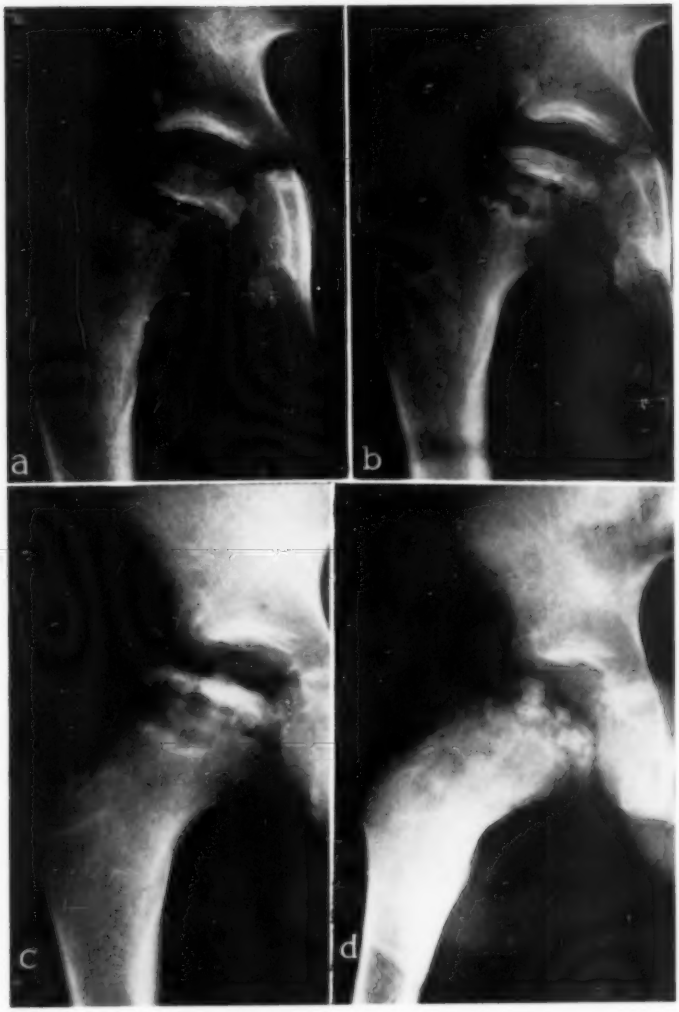


Fig. 1

e . . . Nov. 16, 1931. At eighteen months (one month after operation), no dense areas are seen. The head is filling in and is rounder, but irregularly ossified. The neck is growing into the head. The capsule is not swollen.

f . . . Feb. 8, 1932. At twenty-one months (four months after operation), more filling in is seen. Ossification is more even. Half of the head is outside the acetabulum.

g . . . Nov. 5, 1932 (thirty months after onset and thirteen months after operation). The head is more evenly ossified.

h . . . Apr. 8, 1933. Three years after onset and eighteen months after operation, further ossification of the head is shown.

i and *j* . . . Sept. 15, 1934 (four years after onset and three years after operation). The head is well ossified, but flat, thin and broad. The neck is broad and has grown into the head. The course was probably shortened by drilling, but drilling might have been done earlier with more benefit. Apr. 19, 1947 (seventeen years after onset and fifteen and one-half years after operation). The head is rounded, but lipping appears at each margin. One third of the head is outside the acetabulum.

k . . . At operation on Oct. 22, 1931, the synovial membrane showed marked edema and hypervascularity. Villi are present. There is marked infiltration with plasma cells and lymphocytes. (Author's article in The Journal of Bone and Joint Surgery, Vol. 30-A, published by The American Orthopaedic Association, Inc.)

coxa plana by the history, acute illness with fever and acute signs, laboratory tests indicative of infection, and a much more rapid course in the roentgenograms without the usual features of coxa plana. Similarly osteomyelitis, rheumatic fever, and Still's disease should be easily distinguished. Slipping of the upper femoral epiphysis in the early stage is clinically undistinguishable from coxa plana, but the roentgenographic features and the age incidence are distinctive.

COURSES IN UNTREATED CASES

Coxa plana follows a characteristic course, growing progressively worse for about two years, then progressively heals. Although there is considerable variation between individual cases the basic features tend to run true to form. In studying the results of treatment the natural course of the disease and its basic variations must be kept in mind, as well as the variations in the time table. The disease always heals in time, and the results are not bad, at least in childhood, with no treatment at all.

TREATMENT

At the present time the basic treatment for coxa plana is rest. Rest for the hip may be most easily obtained in this age group by rest in bed. However, rest must be continued for two or three years from the onset in the average case. There should be no weight bearing, even on kneeling. Motion is desirable. Rest favors recovery of the circulation of the soft tissues and healing of the bone and prevents further damage by weight bearing and activity. Rest is most important in the early stage when the disease may even be avoided by prompt treatment, and during the period when the decalcification of the ossification center is marked. If pain and spasm are acute, traction for two or three weeks is desirable. The hips should be maintained in some flexion, at least the major part of the day, in

ROENTGENOGRAMS—FIG. 2

Fig. 2. Typical case of coxa plana, in a child of 4½ years, treated with a sling and crutches. An early diagnosis was made. Pain and limp had been present for three weeks. All motions were limited by pain and spasm. Healing was not complete after four years without weight bearing. In this instance the lesion is seen better in lateral views.

a . . . Oct. 20, 1943. The capsule is swollen. The head is smaller and thinner than normal, irregularly ossified, and the articular surface is uneven. The joint space is wide, superiorly and medially. The patient was kept in bed for two months, then a sling and crutches were used.

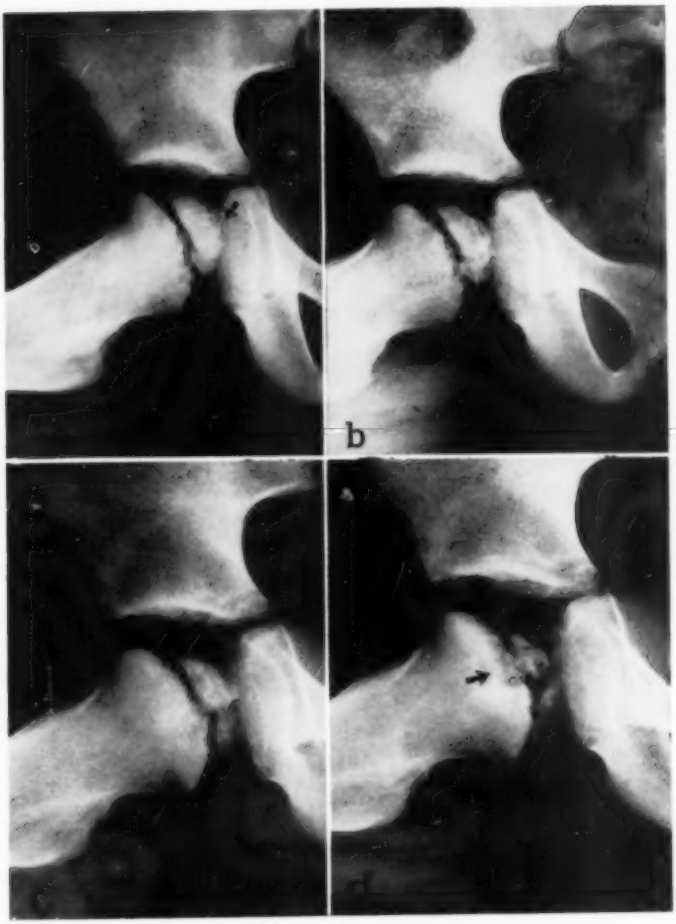


Fig. 2

b . . . Mar. 29, 1944. Five months after onset, ossification of the head is more irregular and dense. The epiphyseal line is wider, with decalcification at the junction with the neck.

c . . . Oct. 2, 1944. After one year, ossification of the head is more irregular, especially posteriorly. The head is more dense centrally. The neck is wider. The epiphyseal line is healing. The proximal surface of the neck is convex, growing into the head.

d . . . Jan. 3, 1945. At the end of fourteen months, ossification of the head is more irregular, with absorption anteriorly. The head is flat anteriorly, thin and broad. The epiphyseal line has healed.

TABLE. CHRONOLOGIC HISTORY OF COXA PLANA

Early Clinical Features (from onset thru 1-2 years):
- Protective limp.
- Pain, referred to groin, inner side of thigh or knee.
- Motions slightly limited, with pain and spasm at extremes.
- Atrophy of thigh.
- Slight elevation of erythrocyte sedimentation rate.

Early Pathology (Few months to 2 years):
- Synovial membrane swollen, edematous, vascular, villous.
- Lymphocytic infiltration, especially about the blood vessels.
- Scattered plasma cells and mononuclear wandering cells (histiocytes).
- Degeneration of hyaline cartilage of epiphyseal plate.
- Fibrocartilaginous repair at junction of plate with neck.
- Bone trabeculae of capital epiphysis irregular in architecture.

Early Roentgenographic Changes:
- First few weeks: Swelling of joint capsule.
- First few months: Flattening or irregularity of articular surface of ossification center of capital femoral epiphysis, usually laterally, with widening of joint space superiorly.
  - Cartilaginous head remains round.
- Neck begins to widen.
- Widening of epiphyseal line, with decalcification at its junction with the neck.
- Widening of joint space inferiorly, due to swelling and thickening of soft tissues at haversian gland.
- Increased density of a part of the ossification center, usually the central portion.
- Areas of diminished density in the ossification center, usually medially and laterally
- Second year: Epiphyseal line begins to recalcify.
  - Decalcification of the dense area, which breaks up into small bone islands surrounded by granulation tissue.
  - Recalcification of decalcified areas at margins of head.
  - Proximal surface of neck becomes convex, growing into head.
  - Epiphyseal line reossified and healed.
  - Swelling of capsule subsides.
- Third year: Recalcification of central portion of head begins.
  - Head wide, thin, rounded, but much flatter than normal and lateral third projects beyond acetabular margin.
- Third to sixth year: Acetabulum begins to conform to shape of head.
  - Femur short, due largely to flattening of head and shortening of neck.
  - Ingrowth of bone from neck into central portion of head not uncommon.
  - Head fills in with bone, but texture and density are uneven.

Late Clinical Features (3 or more years after onset).
- Limp, due to shortening and mechanical limitation.
- Motions slightly limited, especially abduction and rotation, by incongruity of femoral head and acetabulum, and by contracture of the capsule and ligaments.

Late Pathology:
- Synovial membrane thickened, avascular and sclerotic.
- Capsule thickened and contracted.

Late Roentgenographic appearance (5 or more years after onset):
- Sixth to seventh year: Head completely filled in, texture homogenous. Head and neck broad, head thin, flattened, and projects beyond acetabulum about one third.
- Osteoarthritis, with lipping at margins of head and acetabulum, thinning of joint space with condensation of adjacent bone, occasional cystic degeneration.

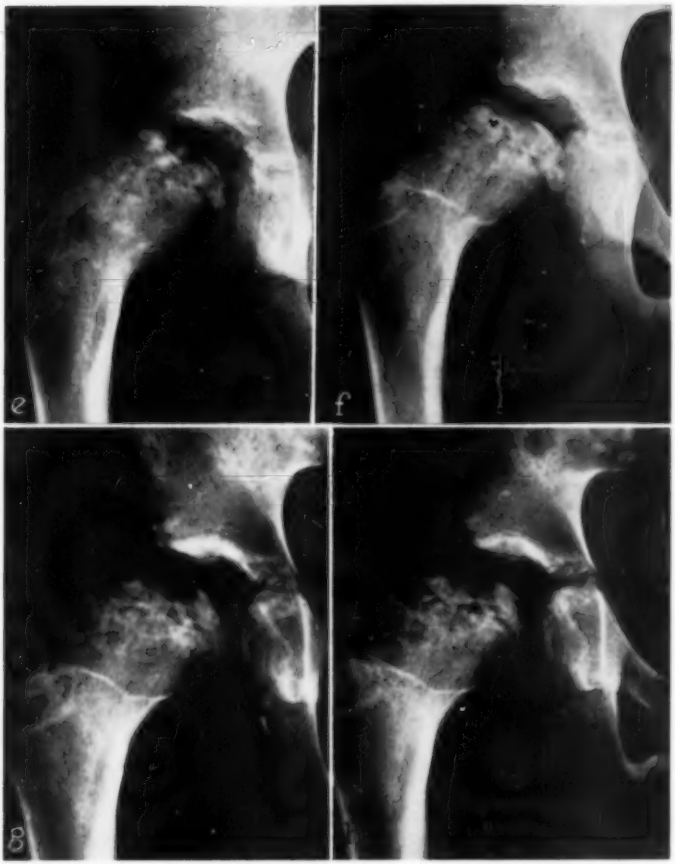


Fig. 2

e . . . Feb. 13, 1946. After twenty-eight months, the anterior half of the head has been absorbed, except for a small island; the posterior half is ossifying and filling in. This is the *end of the absorptive phase.*

f . . . Sept. 23, 1946. After three years, slightly more filling in is seen. Weight bearing is begun.

g . . . By Oct. 8, 1947, the posterior half is nearly healed, but there is no filling anteriorly. Compare with anteroposterior view.

h . . . The head is broad, thin and flat. The neck is broad. Slight subluxation has occurred. (Author's article in The Journal of Bone and Joint Surgery, Vol. 30-A, published by The American Orthopaedic Association, Inc.)

order to relax the soft tissues at the hip and allow more freedom for the circulation. Rest should be continued until all pain and spasm have subsided, and until there is sufficient regeneration in the head for safe weight bearing.

The best ambulant treatment for coxa plana is the use of an ankle sling attached to a belt of the Sam Browne type, with crutches and no weight bearing on the affected leg. However, this method is difficult for little children. A leg brace may be used, with the affected leg dangling and the opposite shoe built-up, but such a brace is clumsy for young children and maintains the hip in extension instead of relaxed in flexion. Crutches alone may be used, but young children cannot ordinarily be depended upon to avoid weight bearing, and some of them find crutches difficult. Gentle swimming is not only permissible but desirable.

Immobilization of the hip in a plaster cast is undesirable as it is likely to result in permanent limitation of motion and does not favor restoration of the circulation. Similarly, continuous traction over a period of time is undesirable.

After the ossification center has regenerated and the lesion is healed, weight bearing is begun gradually, with restriction of vigorous activity for several months longer. While weight bearing is being resumed the hips should be observed closely clinically and roentgenographically. Should symptoms or signs recur, or the roentgenogram reveal unsatisfactory progress, weight bearing should be delayed.

ROENTGENOGRAMS — FIG. 3

Fig. 3. *a* . . . The pelvis in Morquio's disease, or deforming osteochondrodystrophy. Here the head of the femur is not yet ossified, but when ossification occurs the femoral head is large, the neck broad and short, the acetabulum shallow, and the head and acetabulum are deformed. While the femoral head may resemble coxa plana the total picture is quite different.

b . . . Cretinoid epiphyseal dysgenesis. Ossification is appearing in the head of the femur in an irregular manner, not unlike that seen in true coxa plana and in similar disorders. The neck is broad, apparently by extension medially, and the capital epiphysis is at the top or outer side of the neck. The acetabulum is enlarged, shallow, and the superior margin oblique, all more pronounced than would normally be seen in simple coxa plana.

c . . . Coxa plana changes in congenital dislocation of the hip, reduced by manipulation one year previously, position maintained in a double hip spica with the hip in abduction and internal rotation, thus affecting the circulation to the epiphysis. There is yet slight subluxation, the acetabular roof is oblique, the head is flat, shallow, and irregularly ossified, the neck broad. This complication of the treatment of congenital dislocation of the hip is *usually symptomless* (perhaps because the hip is usually immobilized and the baby not bearing weight on it when it occurs), tends to head promptly (perhaps because of the younger age), but there is usually a residual deformity with incongruity between head and acetabulum which leads eventually to osteoarthritic changes.

Fig. 3 A

Fig. 3 B

Fig. 3 C

Fig. 3 D

d . . . Congenital subluxation of hip at age 45 years. The femoral head is broad, flat and mushroomed, the neck short, with moderate anteversion. The acetabulum is flattened, shallow and elongated. There are marked osteoarthritic changes, with thin joint space, irregular condensation of the cortical bone, and cystic degeneration of the acetabulum and femoral head.

OPERATION

An effort has been made to restore circulation to the femoral head more rapidly by drilling holes across the epiphyseal plate. It has been found that the operation reduces the period of the healing by twenty-five to thirty-five percent, and that the residual deformity is usually less. However, the course of the disease is still much too long. Another operation is being used now but it is too early to report results, although the preliminary report is encouraging.

DISCUSSION

Much has been learned in the past four decades regarding the clinical and roentgenographic course of coxa plana, and the pathology has been studied. The disease is known to be a vascular disturbance of the capital epiphysis of the femur and to run a self limited course. However, the exact cause of the vascular disturbance is not known and no rapidly effective treatment is available. It appears that very early recognition of the synovitis which precedes the development of coxa plana, with prompt institution of non weight bearing may result in the abortion of at least some of the cases. This is our best preventive measure at the present time. When more is learned of the cause of the disease, prevention should be even more effective. The treatments used at the present time consist basically of preventing damage to the hip while the disease runs its course and regeneration occurs. A more effective treatment should be sought, especially one which can reduce the period over which the hip must be protected. Herein lies the future for progress in this field.

REFERENCES

Calvé, J.: Sur une forme particulière de pseudo-coxalgie greffée sur des dèformations caractéristiques de l'extremite supérieure du fémur. Rev. de Chir. 30:54, 1910.
————: Osteochondritis of the upper extremity of the femur. J. Orthop. Surg. 3:489, 1921.
Danforth, M. S.: Treatment of Legg-Calvé-Perthes' disease without weight bearing. J. Bone & Joint Surg. 16:516, 1934.
Ferguson, A. B., and Howorth, M. B.: Coxa plana and related conditions at the hip. J. Bone & Joint Surg. 16:781, 1934.
Gill, A. V.: Legg-Perthes' disease of the hip; its early roentgenographic manifestations and its cyclical course. J. Bone & Joint Sur. 22:1013-1047, 1940.
Goff, Charles C.: Legg-Calvé-Perthes Syndrome. Charles C. Thomas, Springfield, Ill., 1954.
Howorth, M. B.: The osteochondroses. Advances in Pediatrics, Interscience 3:298, 1948.
————: Coxa plana. J. Bone & Joint Surg. 30A:601-620, 1948.
Howorth, M. B.: Textbook of Orthopedics, Philadelphia, W. B. Saunders Co., 1952.
Legg, A. T.: End results of coxa plana. New York, J. Bone & Joint Surg. 9:26, 1927.
————: An obscure affection of the hip joint. Boston Med. & Surg. Jour. 162:202, 1910.
Lewis, R. W.: Correlation of the roentgen and pathologic findings in Perthes' disease. Radiology 22:188-190, 1934.
Perthes, G. C.: Uber arthritis deformans juvenilis. Deutsch. Ztschr. f..Chir. 107:111, 1910.
Waldenström, H.: Coxa plana, osteochondritis deformans coxae, Calvé-Perthessche Krankheit, Legg's disease. Zentralbl. f. Chir. 47:539, 1920
————: First stages of coxa plana. J. Bone & Joint Surg. 20:559, 1938

As readers probably noted, the December issue of *Archives of Pediatrics* republished Dr. Louis Fisher's fifty-year pediatric progress report—1884-1934. Now marking its 75th Anniversary, this journal is pleased to present Dr. Bass' comprehensive review of the last twenty-five years.

PEDIATRIC PROGRESS DURING THE LAST TWENTY-FIVE YEARS

MURRAY H. BASS, M.D.*

It is always a pleasure and a great satisfaction when one can write of the successful outcome of some problem such as the correct diagnosis and cure of an unusual and obscure disease, or of the correct solution of some difficult experiment. In the same way, it is really a thrilling experience for a pediatrician to look back over a span of twenty-five years and note the enormous advances that have been made in his chosen field. With the possible exception of atomic physics, no science has made greater progress in the last quarter century than has medicine. Since pediatrics has so much in common with general medicine, it has had its full share in this extraordinary advance. The discovery by Domagk in 1935 of the therapeutic value of prontosil leading to the development of the sulfa drugs caused as abrupt a change in medical care as did, in years gone by, the great discoveries of anesthesia, antisepsis, the x-ray, salvarsan or insulin. When penicilin and the mycin drugs were also added to our armamentarium, the entire aspect of the treatment of children's illnesses completely changed. A great many of the cases the pediatrician has to deal with are acute infections due to micro-organisms sensitive to these drugs, and as is now so well known, illnesses which might last many weeks were abruptly cured and many which were almost uniformly fatal, are now curable.

Twenty-five years ago the pediatrician carried in his bag a syringe for chest aspiration for diagnosing empyema, which was a very common ailment. Now empyema is a relatively rare disease.

In the cold months of the year, hardly a day went by that the pediatrician did not see a case of otitis media which, in many instances, went on to suppuration so that on a busy pediatric hospital service, there were always cases of mastoiditis, some with intracranial complications such as lateral sinus thrombosis and sepsis,

* *Consulting Pediatrician, Mt. Sinai Hospital, New York City.*
Late Associate Clinical Professor Pediatrics, Columbia University.

or perisinus or cerebellar abscesses. The pediatricians were quick to realize that the sulfa drugs if given early and long enough would prevent these complications and cure the otitis. The otologists, many of whom did not agree with this and were afraid of masking the disease, discontinued the drugs too soon. I recall a meeting at which an important otologist ended a paper on the subject with the words "The outlook for the sulfa drugs is very black indeed". After the meeting, one of the pediatricians present remarked that he thought "the outlook for the *otologist* is very black indeed!" And he was right, for mastoiditis, with its serious complications, has become a very rare disease.

Another example is the change in the outlook in meningitis. When due to hemophilus influenzae the outlook twenty-five years ago was practically 100% fatal. Today, the fatality rate is about 5%, though 10 to 15% may show some permanent cerebral damage.

Streptococcus infection, scarlet fever, erysipelas and many cases of osteomyelitis were a terrible worry for the pediatrician, who twenty-five years ago, had only palliative and symptomatic remedies, whereas now, he has specific treatment easily available and surgery is much less frequently needed.

During these years, we not only have a large number of potent antibacterial drugs from which to choose, but we have tests for the sensitivity of these drugs which permit us to avail ourselves of the most suitable drug for our particular case.

This is but one example of how great a role the laboratory has gradually assumed in the last three decades in determining the diagnosis and treatment of patients under our care. The necessity of greater knowledge of the basic sciences, in order to understand and conduct these laboratory techniques has greatly changed the curriculum of the medical student, with the result that the recent graduates in pediatrics have a better scientific background than the practitioners of 25 years ago, and often know more actual scientific medicine than the older men. As a matter of fact, the speed with which new knowledge concerning pediatrics is being acquired raises the question as to whether it may not finally be necessary for the average children's physician to specialize in one phase of pediatrics. Thus we are finding it extremely useful to have pediatricians who are specializing in pediatric allergy or pediatric renal disease, or pediatric cardiology. Even at the present time, only the most highly intellectually-gifted man can properly conduct a pediatric hospital service without the assistance of others to whom he can turn for special advice.

In 1941 an Australian oculist, N. McAllister Gregg, noted that he was seeing an unusually large number of congenital cataracts in newborn infants and made the clever observation that the mothers of these children had suffered from rubella early in their pregnancies. He also pointed out that many of these infants showed other congenital defects, such as deafness, glaucoma, cardiac malformation and mental retardation. These findings were corroborated in many parts of the world and led to the conclusion that congenital defects, which had, for the most part, been attributed to hereditary genetic factors, could be due to environmental effect on the developing fetus. Other investigators, especially Joseph Warkany, had been describing fetal malformations in animals due to dietetic deficiencies in the pregnant mother. Studies such as these together with the discovery of the rubella syndrome in human newborns led to a search for other diseases of the pregnant woman affecting the fetus, and, as we shall point out in a discussion of virus diseases, moreover it became evident that environmental factors were of extreme importance.

In the meantime, the study of genetics underwent considerable progress and diseases due to actual change in the genes were more carefully studied. It has been possible to show that the cause of a hereditary disease in an infant may be due to the absence or malfunction of a single enzyme and that this difficulty resulted from the malformation of a single gene. An exquisite example of this is the inborn error of metabolism in chronic galactosuria. The clinical manifestation of hepato and splenomegaly, malnutrition and cataracts had been found to be due to the presence of large amounts of galactose in the blood and that when galactose-free diet was prescribed for such infants, they improved. Now, however, with the newer knowledge, we know exactly where the fault lies and that the biochemical defect lies in the faulty action or absence of the enzyme which converts galactose-1-Po_4 to glucose-1-Po_4. The disease thus results in hypoglycemia and excess of galactose, a substance toxic for the body.

Another field in which clinical disease and its genetic origin has been explored is anemia due to various types of hemoglobin. For many years methemoglobinemia had been known as a congenital illness due to a peculiar form of hemoglobin. But with the discovery of paper electrophoresis it was found that there were many varieties of hemoglobin. In 1949 it was shown that sickle-cell anemia was due to an abnormal type of hemoglobin designated as Hemoglobin S and that the sickle-cell "trait" and sickle-cell "disease" differed as

the result of a genetic variation. "The ability of the body to form the abnormal hemoglobin appears to be determined by a mutant gene. If that gene is represented but once, some 20 to 40% of the hemoglobin is of the abnormal type and a trait or carrier condition results. When the gene is represented twice, in consequence of having been inherited from both parents, virtually all of the hemoglobin is of the abnormal type, and a hemolytic anemia usually results." [1] Up to the present, at least eight varieties of hemoglobin have been described, among them, Hemoglobin F found in the human fetus.

How has this vast amount of knowledge relating to pediatrics that has been accumulating at faster and faster rates in the last few years been brought to the attention of the individual physician? The busy practitioner has only a limited time to read, but we are fortunate to have more hospital conferences and medical meetings at which this knowledge can be disseminated. Of special importance in this field, is the American Academy of Pediatrics. Organized in 1930, the society has grown by leaps and bounds. With an attendance of 304 men at its first meeting in 1931, it has grown to its present membership of almost 6000. In 1933, the American Board of Pediatrics was founded and has continued its work to date, so that to call himself a specialist in pediatrics, or to become a member of the AAP, a physician must have passed a rigid examination. This has undoubtedly raised the standard of pediatric medicine in the United States both in hospitals and in private practice, and must incidentally have stimulated men to do better work and to help improve child health in all its phases. The booklets which each member receives from time to time on important advances in pediatrics (contagious diseases, care of the newborn, etc.) are most authoritative and helpful, as are the panels and round-table discussions on many special subjects to which members of the Academy may subscribe.

It is only relatively recently that the surgery of children has become a special branch of general surgery. With the many advances in the diagnosis of conditions in infants requiring surgical care, surgeons particularly conversant with such diseases, especially with congenital malformations, became a real necessity. Pediatric surgery, dealing as it does, with very delicate tissues and with very small fields of operation, requires special knowledge, special training and special techniques. Those of us who have been in pediatrics a long time, must agree that since we have specially trained surgeons we see better results. However, it is not only the man who does the actual operating who is responsible for the greater numbers of

recoveries, it is also our great advance in our knowledge of the preparation of the patient for operation,—in hydrating the infant, in choosing the right anesthetic, and in conducting correctly the postoperative care. For example, a condition such as tracheo-oesophageal fistula or oesophageal atresia in the new-born can now be relieved by anastomoses of the upper oesophagus with a portion of the ascending colon with excellent results.

Not only has technique been perfected, but new causes of disease yielding to surgery have been discovered. As an example, one might cite Hirschsprung's disease (megacolon) which we now know is due to absence of ganglion calls in the lower end of the colon with secondary distension of the proximal gut. By removing the affected gut and bringing the healthy gut down to the anus, these cases are cured.

The outlook in operations in young infants and newborns has become so much better that diseases such as pyloric stenosis are referred for the Ramstedt operation much oftener than formerly. I believe that most of these cases if medically treated early and vigorously, can be cured; but the course is a very long and difficult one. With modern surgery, the obstruction is quickly removed and the infant leaves the hospital well in a few days' time, and the recovery rate is almost 100%.

With the improvement of pediatric cystoscopes and the introduction of pyelography and the urinary antiseptics, the outlook for congenital anomalies of the urinary tract has also greatly improved.

Relatively recently, the entire world has become interested and astounded at the development of cardiac surgery, where twenty-years ago, we had to be satisfied with the diagnosis of "congenital heart disease" for which nothing could be done, we are now able to diagnose and remedy many of the various lesions. In 1938 Gross in Boston successfully tied off a persistent ductus arteriosus. In Baltimore, Blalock and Taussig in 1945, reported the restoration of good circulation in cases of Tetralogy of Fallot. Cardiac catheterization, the study of oxygen saturation in various areas in the heart and great vessels, the improvement in radiography of the cardio-vascular system and recently, the development of pump oxygenators to bypass the circulation in the heart itself, and finally the use of hypothermia, have all contributed to this amazing advance.

Not only the heart itself, but disease due to anomalies of the great vessels are now much easier to diagnose and also yield to surgical treatment. A case in point is that of a young infant brought on account of great difficulty during feeding when she choked and

became so cyanotic that the parents would hold her outside the window in the hope that the fresh air would help to avoid asphyxia. Radiography revealed an aberrant vessel crossing the trachea and oesophagus. At operation, the vessel was divided and the infant made a perfect recovery. Twenty-five years ago, neither the diagnosis nor the cure would have been possible.

Not only the surgical, but also the medical treatment of cardiac disease in childhood has undergone great change in the past few years. Rheumatic heart disease originating, as it does, as a result of streptococcal disease, has decreased with the introduction of antibiotic prophylaxis. A news release by the American Heart Association in 1958 has this to say on the subject: "The death rate from rheumatic fever and rheumatic heart-disease among young people, five to twenty-four, has reached an all-time low. Although the rate has been declining continuously over the entire period since national vital statistics have been available, the decline has been much more pronounced since about 1945 when penicillin became generally available. In this brief period of ten years, the rate has dropped 70%."

The value of the steroids versus the salicylates in the treatment of the acute stage of rheumatic carditis is still under discussion.

Of all the advances that we have seen during the past twenty-five years none is of more significance than the discovery of the Rh factor in human blood and its effect on the fetus and newborn infant. During the third decade of the century, many papers appeared dealing with obscure transfusion reactions which led to the supposition that the blood of certain individuals must contain substances harmful to others. The blood groups A, B, AB and O had been discovered early in the century, but it was not until 1941, that the discovery of the Rh factor in human blood became of clinical importance. Not long before that, what we had previously called congenital anemia or icterus gravis, or fetal hydrops, were shown to be different manifestations of the same illness,—erythroblastosis fetalis, caused by incompatability of the child's and mother's bloods. The introduction of the Coombs test in 1945 made the diagnosis simpler and more certain, while the introduction of exchange transfusion at about the same time brought about the saving of many desperately sick infants.

During the period of time under discussion, several heretofore unknown diseases have been described. Among these may be mentioned Cystic fibrosis of the pancreas; Infantile cortical hyperostosis (Caffey's disease); Familial autonomic dysfunction (Riley-Day syndrome); Retrolental fibroplasia; Hyaline membrane disease;

Cytomegalic inclusion body disease; Agammaglobulinemia; Toxoplasmosis. Coincidental with these discoveries a considerable number of new clinical entities have appeared due to the newer knowledge of viruses. As a matter of fact, virology has become more and more important. And due to the improvement in this field, the etiologic agents causing many previously-described diseases, have been discovered,—among them may be mentioned poliomyelitis, mumps, influenza and new groups such as the ECHO viruses, Adeno viruses, Coxsackie viruses and Herpes viruses. The ability to grow the viruses or to study antibodies produced by them has solved many problems leading to greatly improved diagnosis and therapy. To mention a few: the discovery of the three types of viruses causing poliomyelitis and the production of the Salk vaccine; the recognition of disease resembling poliomyelitis, but due to Coxsackie viruses; the many gastro-intestinal and respiratory ailments, the causes of which have been established. Special mention should be made of the value of noting the increasing titre of virus antibodies as an aid to diagnosis which has been so helpful in the nonbacterial encephalitides such as that due to mumps.

As was mentioned, the fetal damage due to rubella, stimulated investigation of other diseases of the pregnant woman which may affect the developing fetus. We have recently learned that besides the fetal damage due to maternal mumps, measles, varicella and variola, other viruses, such as herpes simplex, cytomegalic inclusion disease, poliomyelitis and Coxsackie virus, may transplacentally reach the fetus and cause disease, often even if the mother shows no overt symptoms of illness.

It is of interest to note that while new clinical pictures are presenting themselves some diseases are gradually disappearing, since their etiological cause has been discovered. We might mention retrolental fibroplasia which has been found due to excessive oxygen given to newborns. The cause of acrodynia, so long a baffling problem, has been solved and we now know that in most cases, the disease is a manifestation of mercury toxicity and that the illness frequently followed the use of teething powders containing calomel. In the future, we will surely see fewer cases of this frequently fatal disease.

Our attitude to tuberculosis in infancy and childhood has undergone a complete change, for instead of merely diagnosing this disease, we now have specific drugs which even arrest and cure such a previously hopeless condition as tuberculous meningitis.

Tuberculous adenitis and bone involvement have diminished in number and severity and are now unusual illnesses. In this connection, the statistics from Bellevue Hospital Children's Service over a period of twenty years, published by Edith Lincoln, are pertinent: in the pre-chemotherapy period between 1930 and 1946, the mortality due to primary tuberculosis was 21.5%. During the next four years, when streptomycin, para-aminosalicylic acid and promisole were used on only 35% of the patients, the mortality was reduced to 5%. Upon the addition of isoniazid to the treatment in 1952-53, the mortality fell to 1.5%.[2]

Among the great advances of late years is our use of parenteral therapy and the regulation of electrolytes. The introduction of parenteral fluid, amino-acids, vitamins and plasma in the treatment of diarrhoeal diseases resulted in a great drop in their mortality and the relatively recent contributions in the importance of electrolytes has still further reduced the death rate. Much of this is due to the improvement in laboratory methods such as the use of the spectrophotometer which obtains results in a very short time compared with the tedious, time-consuming, complicated methods previously necessary. The importance of potassium in the child's metabolism and the use of the electrocardiogram in determining its concentration in the blood is an interesting and important development.

The fractionation of plasma and the discovery that antibodies are contained in the gamma-globulin fraction has led to its intramuscular use in a number of diseases, particularly measles, which we may modify or prevent.

Interest has been focused on the newborn, so that nurseries are more carefully supervised, the prevention and control of infections are more emphasized, the care of prematures is being improved, and the importance of better records such as death certificates for neonatal deaths is being urged. Cooperation between hospitals in various parts of the country are studying various aspects of this problem, as for example, the ten-year prospective study of the cause of congenital anomalies just now being launched in St. Louis. The importance of such research may be shown by the fact that in New York City, 16.5% of the deaths of infants under one year and 14% in children age one to fourteen years, have been due to congenital malformations.

One field in which a great deal has been accomplished in recent years, but one in which there is much still to be done, is that of perinatal mortality. In this the medical school, the obstetrician, the

pediatrician, the pathologist, the social-worker and the department of health all must collaborate. During the past decade we have come to realize that by such cooperation, coupled with the advances in the obstetric and pediatric care, we can continue, and even improve on the remarkable saving of infant and maternal lives as shown on the accompanying table.*

Progress in Saving Lives of Mothers and Babies in 50 Years

Infant Mortality Rates Under 1 Year (per 1000 live births)	1908	(1910)	1957	Percent Change
TOTAL	111.2	113.4	25.0	−77.5
Under 1 Month		40.8	19.2	−52.9
Postneonatal — 1 Month to 1 Year		72.6	5.8	−92.0
Puerperal Deaths				
Number	699		107	
Rate per 10,000 live births	47.9		6.4	−86.6
Deaths By Age — Numbers				
Under 1 Year	16,231		4,176	
1 Year	4,231		243	
2 Years	1,789		145	
3 Years	1,122		117	
4 Years	768		86	
Total Deaths Under 5 Years	24,141		4,767	
Population Under 5 Years	485,000		677,000	
Rate per 100,000 population (Under 5 Years)	4,977		704	−85.9

* I am indebted to the New York City Health Department Bureau of Records and Statistics for permission to use this table.

In the foregoing review, it has been possible to mention only some of the most important advances that have taken place, and most of these have been concerned with the cause, recognition and treatment of organic disease. Many important items had to be omitted for the sake of brevity, among them the antifolic drugs, the use of steroids, the abolition of gonorrheal infection, especially vaginitis, and the reduction to a rarity of congenital syphilis by the use of penicillin and premarital Wassermann tests. However, during the period of time we are discussing, there has been going on a great change in the actual practice, in contrast to the science, of pediatrics. The newer knowledge in the field of psychology has had a great influence on the care of children and the whole outlook of the pediatrician has changed. Where previously, he took care of the illness, he now takes care of the child. Psychical trauma has been recognized as being as important to the future adult as physical trauma. We now prepare the child for operation not only with physical remedies, such as enemata or diet restriction, but also with careful attention to the psychic disturbance which may follow hospitalization. Recovery

rooms are found in many hospitals where the child, when coming out of anesthesia will find his mother, rather than a stranger with him, surrounded by the bare walls of a hospital ward. This concept of the treatment of the whole child has led the pediatrician to study and advise about the relation of the child to his parents, his siblings, his friends and his school. This is partly the result of the introduction of periodic examinations when the physician sees the child when he is well and can note the state of his growth and development—mental, emotional and physical. From this it also follows that the relation between the child and physician becomes a much closer one. The following incident is a pertinent example: A mother brought her five-year-old boy to the pediatrician for a routine check-up and said "Here is your doctor." To which the boy replied, "He's not my doctor, he's my friend." The child thus may become the bond between the physician and the parents. And this allows the doctor to become a trusted advisor in guiding his patient from infancy through adolescence.

Whenever an important new, successful method of treatment or prophylaxis is reported, one always hears the practitioner complain that gradually there will be less and less work for him and he will not have enough to do. However, that has not been the case, for as we have noted, there are constantly new fields opening up and new diseases being described and though there may be fewer long-drawn-out "big" cases which were previously so common, there seems to be enough to keep pediatricians as busy as they were twenty-five years ago.

Another complaint that is heard relates to the fact that so much is done in hospitals and by laboratories, that the old type clinician is passing away. On the contrary, careful physical examination of the patient and the wise conclusions to be drawn from the physical findings and the history of his case, are still just as valid in diagnosis as they ever were. An excellent example of this is well described by Charles May in reference to the sudden appearance in certain areas of the country, of repeated convulsive seizures in young infants, later found due to the fact that they were all being fed a propietary food, a change in the preparation of which resulted in the complete removal of vitamin B-6. May writes: "The original observation which called attention to the problem of unusual convulsions in infants and pointed the way to its solution, were made by a doctor in practice, just as were similar observations which led a few years ago to an appreciation of the circumstances, producing a deficiency of folic acid in infancy. This should encourage practitioners to real-

ize that close observation of their patients still permits fundamental contributions to be made without the paraphernalia of a hospital research center. It should also remind professors that conspicuous phenomena are still passing before them unseen." [3]

Those interested in the present state of pediatrics should read the article entitled, "Pediatrics in Transition" by G. M. Wheatley,[4] from which I quote: "In essence, the dissatisfactions with pediatric practice lie more with the individual's own misconceptions of the reality of pediatric practice today, and with the individual's attitude toward children. As Powers has expressed it, 'There are many aspects of pediatric experience which attracts graduates in medicine but I think the chief magnet is the enjoyment and satisfaction of working with and for children—not sick children, not healthy children, not handicapped children, not infants, toddlers, teen-agers or adolescents,—just children!' "

REFERENCES

1. Neel, James V.: Human Hemoglobin Types. N. E. Journal Med., 256:161, 1957.
2. Linoln, Edith: The Effect of Antimicrobial Therapy on the Prognosis of Primary Tuberculosis in Children. Am. Rev. Tuberc., 69:682, 1954.
3. May, Charles D.: Proc. Inst. Med., Chicago, 20:83, 1954-5.
4. Wheatley, G. M., J.A.M.A., 168:856, 1958.

1112 *Park Avenue, New York City*

Clinical Conference

THE MT. SINAI HOSPITAL, New York

June 12, 1958

CONGENITAL CEREBRAL ANEURYSMS AND COARCTATION OF THE AORTA

HORACE L. HODES, M.D.,
LEONARD STEINFELD, M.D. AND SIDNEY BLUMENTHAL, M.D.

DR. HODES: This is a child who came to our Cardiac Clinic. He has been seen there by Dr. Steinfeld and by Dr. Blumenthal, who will take part in the discussion.

The child's history will be presented by Dr. Warren.

DR. WARREN: This is the first Mt. Sinai Hospital admission of a 14 year old male child who is a known cardiac patient since age 6 years when, during a routine school physical examination, a murmur was heard. He subsequently was referred to the clinic at Mt. Sinai where a diagnosis of coarctation of the aorta was made. The possibility of associated sub-aortic stenosis was also entertained. There were no symptoms whatever in this child. Blood pressure during the clinic visits was 160/100 in the arms, but no blood pressure was obtainable in the legs.

Surgery was advised by the Cardiac Clinic but was initially rejected by the parents. When the parents finally agreed, at one point the child was scheduled to be admitted, but he ran away because he was frightened.

The present illness began approximately one day prior to his admission to the hospital. During that afternoon in school, he was observed by his teachers to be somewhat confused; during an examination in the school—he walked out of class.

On the way home from school, he was seen to faint. He was observed to be unconscious, but there were no specific observations as to whether he had a seizure at that time. He was taken to another hospital where he was found to be quite drowsy. He complained of headache. On two occasions at that hospital, he vomited. He was kept there overnight and the next day was transferred to the Mt. Sinai Hospital, when it was learned that he was a clinic patient here.

The past history was essentially unremarkable except as previ-

ously mentioned. There were no previous syncopal episodes. There was no shortness of breath, no ankle edema; there was no history of chest pain. At three years he had chicken-pox and at seven years, he had measles.

Physical examination on admission revealed a well-developed boy who appeared somewhat drowsy; however, he did answer questions and was fairly cooperative. Blood pressure in the right arm was 146/90; in the left arm, 144/90. Blood pressure in the legs was not obtainable. Temperature was 99.6, pulse was 100 per minute. Respiratory rate was 20 per minute and respirations were unlabored.

There was no enanthem. Examination of the head revealed no masses or areas of tenderness. Eyes: conjunctival and sclerae were normal. Pupils were equal and reacted well to light; extra-occular movements were intact. Fundi were well visualized and were normal. Ears and nose were normal. Pharynx was normal. Examination of the neck revealed mild stiffness on extreme flexion. There was no significant enlargement of lymph nodes. Examination of the chest was normal. Examination of the heart: the point of maximum impulse was in the fifth intercostal space, about 3 cm. beyond the mid-calvicular line. Heart rate was 100 and regular. A thrill was felt in the right side of the neck, just above the clavicle. A grade II harsh basal systolic murmur was clearly audible; it radiated down the left sternal border and was well heard in the back. Examination of the abdomen revealed no masses.

With regard to the extremities, the femoral pulsations were barely palpable on each side. The popliteal and dorsalis pedis arteries were not palpable on either side. There was no edema of the lower extremities and no change in color.

The neck, as previously mentioned, was stiff on extreme flexion. The patient was drowsy but he was cooperative. Cranial nerves were intact. Fundi were normal. Deep tendon reflexes were symmetrically hypoactive. A positive Babinski sign was present on the right. Superficial reflexes were present. There was no apparent deficiency in sensation to pin-prick.

In the course of the next several hours in the hospital, the child became somewhat more sleepy and the neck became more stiff. Lumbar puncture was performed. Grossly bloody spinal fluid was obtained with an opening pressure of 350 mg. of water and a closing pressure of 120. The supernatant fluid obtained by centrifugation was slightly xanthochronic. Only red blood cells were seen on microscopic examination.

Spinal fluid sugar was 46 mg. per cent; protein concentration was 340 mg. per cent. No organisms were seen on direct microscopic examination of the spinal fluid, and cultures were sterile.

The rest of the laboratory work-up was as follows: hemoglobin was 13 grams, white count was 8800 per cu mm. with 75 polys, 6 bands, 6 total lymphocytes, and 4 monocytes. The sedimentation rate was 3 mm. per hour. Blood cultures were sterile. BUN was 10; blood sugar was 65 mgs. per cent; CO_2 22.4 mEq.; chloride 100 mEq., sodium 142 mEq., and potassium 4.2 mEq.

Chest x-ray revealed no evidence of pulmonary disease. X-ray findings with regard to the heart and the ribs will be shown shortly.

Intradermal PPD tests were negative. EEG showed no specific abnormality.

Patient's course in the hospital: the child was put directly to bed and kept as quiet as possible; laxatives were prescribed.

Physical findings during the next few days remained essentially unchanged. The patient remained drowsy and the neck remained stiff and the right Babinski continued to be positive. He took fluids well by mouth. Temperature during the first few days in hospital fluctuated reaching 103°F on the second hospital day. It then receded only to rise again on the fourth and again on the fifth day to 103.8°.

Blood pressure also rose gradually and on the 9th of June, the fifth hospital day, it reached 190/118. The boy was therefore given .1 mg. per kilogram of sarpasil intra-muscularly (a total of 4 mgs.). Blood pressure responded promptly, falling to 150/68 within eight hours.

During the evening of the 9th, the child slept well and blood pressure next morning was 146/70. At this point oral serpasil was begun with the intention of keeping the blood pressure down within the day blood pressure again rose to 162/90. At this point, the patient was given another intramuscular injection of sarpasil. On that evening (the 10th of June), he was observed to be somewhat more drowsy than previously. The Babinski sign which had been present earlier only on one side was now observed on both sides.

It was believed at this point that the boy was probably suffering further intracranial bleeding. Spinal puncture was therefore done and pink fluid was obtained. This fluid contained 5500 red cells per cubic mm., and on centrifugation exhibited a xantho-chromic supernatant.

This morning the child's neck is still somewhat stiff; he is slightly more lucid than he was before and he is more cooperative. He is

taking fluids well. Temperature now ranges between 100.5° and 102°.

Dr. Hodes: There are a number of questions that are going to come up that may be debated vigorously. I should like Dr. Steinfeld to begin the discussion.

Dr. Steinfeld: This patient is quite interesting in relation to several patients who have appeared in the clinic recently with relatively normal blood pressure in the face of fairly clear evidence of coarctation of the aorta. There has been considerable discussion on these cases as to whether surgical repair of coarctation is necessary when blood pressure is nearly normal.

A similar situation has occurred in the patient under discussion, although his blood pressure was slightly elevated. It was 140/90, which is elevated for a child, but not remarkably so. This is hardly the level at which we would expect symptoms caused by hypertension. The child has a ruptured aneurysm. This happening may or may not be related to the hypertension, but I think we must assume that it is.

The statistics are quite variable in the literature as to the incidence of aneurysm associated with coarctation of the aorta. Dr. Robert Gross has stated that cerebral aneurysm is often associated with coarctation of the aorta. Dr. Gross states that about 25 per cent of the cases of coarctation die of complications associated with hypertension. The difficulties caused by hypertension are twofold: one is the dangerous effect of hypertension upon intracranial aneurysms. The other is the role of hypertension in causing congestive heart failure.

A number of patients in Dr. Gross' series (the exact number is not indicated) have had difficulties with cerebral aneurysms. Maude Abbott some years ago reported that ten per cent of patients found at autopsy to have coarctation of the aorta, also had one or more cerebral aneurysms. Similar observations have been made in other clinics.

Coarctation of the aorta has been divided into two groups: the type in which the coarctation is found before the point of entrance of the ductus arteriosus (pre-ductal) and those occurring beyond the ductus (post-ductal type).

The position of the coarctation in relation to the ductus seems to have considerable importance in terms of associated defects and prognosis. The post-ductal type of coarctation was previously known as the adult type. It is the type of coarctation which is compatible with a long life span. Most patients who have this type of

coarctation may go into adulthood and only then develop symptoms. The pre-ductal coarctation (infantile coarctation) is associated with a very high incidence of associated anomalies and a very high mortality rate.

Pre-ductal coarctation (infantile coarctation) has been further subdivided into three groups: in one type there is only a narrowing of the aorta just above the ductus. This type comprises about 40 per cent of the cases of pre-ductal coarctation; another 40 per cent have a long segment of coarctation extending from the ductus to the left sub-clavian artery; in the third variety of pre-ductal coarctation, there is coarctation of a long segment of the aorta, including the aortic arch.

The patients in the third category (coarctation of a long segment including the arch of the aorta) have a very high incidence of associated anomalies. About 40 per cent of such patients have intracardiac anomalies, as compared with only about 16 per cent of patients with the other two types of pre-ductal coarctation.

It has been shown that the reasons that children with pre-ductal coarctation have such a high mortality rate is not solely due to the fact that there is no collateral circulation because the ductus lies below the point of aortic coarctation. Rather, it is due in large part to the presence of additional anomalies in such a high percentage of these cases.

It was previously believed that in preductal aortic coarctation, the fact that the ductus came off below the coarctation and supplied the lower parts of the body with blood prevented the development of collateral circulation. This view is no longer tenable. It has been shown that children who have pre-ductal coarctation have blood pressure levels in the upper extremities that are similar to those of patients with post-ductal coarctation. Furthermore, the blood pressure below the coarctation is also quite similar in the two Groups. There is hypertension above the coarctation and hypotension below the coarctation whether there is pre-ductal or post-ductal coarctation. Hypertension is not necessarily related to patency or lack of patency of the ductus, nor it is related to quantity of blood flow across the ductus into the aorta.

The post-ductal (adult) type of coarctation is relatively simple in terms of diagnosis. The majority of patients are now discovered some time in childhood these days as a result of routine physical examination when it is found that the femoral pulses are diminished or absent. A murmur is usually present, but this is not invariably the case. When present, a murmur is usually heard best at the base

and along the left sternal border and it can also be heard posteriorly. The site of the murmur is in no way related to the site of the coarctation, some murmurs being heard a considerable distance from the area of coarctation. Furthermore, the loudness of the murmur does not parallel the degree of coarctation; a loud murmur may be present, even when there is no flow of blood at all past the constricted area. On the other hand, the murmur may be quite soft and faint in the presence of a small narrowing with considerable turbulence past the coarctation. Most observers feel that the character of the murmur is probably related more to collateral flow, to eddying of blood beyond the coarctation and to other unexplained factors. It is certain that the murmur is not necessarily related to the degree of stenosis.

In the post ductal (adult) type of coarctation the heart is not remarkably enlarged on roentgen examination. In fact most patients with coarctation have hearts of normal size. Those patients who have a very large heart are to be suspected of having an associated anomaly; usually this anomaly is a patent ductus. About 15 to 20 per cent of all patients with coarctation have an associated patent ductus arteriosus. Another 15 per cent have associated interventricular septal defects. Therefore, we may suspect that patients with aorta coarctation who have enlarged hearts may have also either patent ductus or inter-ventricular septal defect.

The electrocardiogram in the post-ductal type of coarctation is usually normal. However, there may be some evidence of left ventricular hypertrophy or strain. Notching of the rib is usually present on roentgenograms in post-ductal coarctation. Notching is related to the collateral circulation and usually appears relatively late in childhood. It has been seen very rarely in infancy, and a small number of children 3, 4, and 5 years of age have shown rib notching but this is quite unusual. It generally appears around 9, 10, or 11 years of age. Rib notching is a characteristic sign late in childhood and in adulthood.

Absence of femoral pulse is a very interesting phenomenon, because it may be present when the pressure in the femoral artery is not greatly reduced. It is believed that the reason the pulses are diminished or absent in coarctation is not so much related to the reduction of blood pressure as it is to the interference with the pulse wave as it is transmitted from the proximal aorta to the distal aorta.

I show you now arterial tracings from a child who had absent femoral pulses who was recently operated on for coarctation of the aorta. The pressure in the upper extremities was about 140 mm. In

the lower extremities the pressure was about 90 mm. as determined by the cuff method. We made simultaneous pressure tracings by introducing needles into the femoral and brachial arteries. We can see that the two pressures were fairly close to one another. Certainly, there was no striking difference. However, it was felt that the pulse wave was interfered with as it was transmitted down the femoral artery and therefore one could not palpate the pulsation in the region of the femoral artery.

As one can see on the tracing, there is actually a small difference between the upper and lower arterial pressures, the pressure in the upper extremity being slightly higher. Normally, as you know, the femoral arterial pressure is 10 or 15 mm. higher than the upper extremity blood pressure.

In coarctation the pressure may be fairly good in the areas distal to the coarctation, as in the tracing which I show here. Nevertheless, we do not perceive a pulse. This also bears upon the hypothesis that hypertension in coarctation of the aorta is related to "ischemia" of the kidney. This theory is probably not correct, at least in the majority of cases, because the kidneys are not found to be ischemic. The mean pressure in the area below the constriction may be fairly good (in the range of 90-100) which is certainly adequate for function of the kidney. Therefore the belief that hypertension in aortic coarctation is due to Goldblatt phenomenon is probably erroneous. The hypertension is, more probably due to mechanical factors,—that is, to obstruction to the passage of blood.

I do not intend to go into the complications which may occur in coarctation of the aorta. I shall reserve the rest of the discussion for Dr. Blumenthal.

DR. BLUMENTHAL: Our patient's case forcefully brings to our attention many of the complications of coarctation of the aorta. It is important to discuss this case from two points of view: one is that we are not accustomed to seeing complications very often among our patients—that is, children. Complications occur more commonly in the young adult age group. Secondly, I think complications have become much less frequent since the advent of surgical correction of this anomaly.

In going over the question of complications, I think it is worthwhile to go back to some data that was available in the pre-surgical days (which was not so very long ago) and in the pre-antibiotic era, which also was not so long ago, at least as some of us measure time.

There are two very good series of cases which review this mate-

rial. One is Maude Abbott's, "Statistics on 200 Autopsies", and the other is "A Group of 104 Autopsies," reported by Reifenstein, Levine, and Gross. These are patients who came to post-mortem with coarctation of the aorta. By analyzing the cause of death in these patients, we obtain very important information as regards the prognosis for coarctation of the aorta before antibiotics and surgical treatment were available.

In Maude Abbott's series, 23% and in the Boston Group 26% of these patients died of incidental causes, i.e. either accident, pneumonia, meningitis or other conditions not at all related to coarctation.

Twenty-three per cent of the patients who succumbed died of rupture of the aorta, 16 to 20 per cent of the patients died of subacute bacterial endocarditis or aortitis. Eighteen per cent in the Boston Group and 29 per cent in Maude Abbott's group, died of congestive heart failure and 10 to 13% died of intracranial vascular hemorrhage.

If you sum up these figures, one sees that one-third of all deaths were due to either rupture of the aorta or rupture of an intra-cranial aneurysm, which represents a very significant total.

Regarding the group who died of SBE, their average age was 31. The only interesting thing I'd like to comment on in this group is that in the great majority of cases the endocarditis was implanted on an associated bicuspid aortic valve. The importance of a bicuspid aortic valve in this complication is noteworthy.

Thirty-two of the patients with coarctation who came to post had normal aortic valves. In this group there were no instances of subacute endocarditis. In contrast, among 62 who were found at autopsy to have bicuspid aortic valves, 14 or 23 per cent had subacute endocarditis. Sub-acute endocartitis may occur or aortitis may occur in the presence of a normal aortic valve, but the statistics reveal a very much greater danger to the patient who has an abnormality of the aortic valve in addition to aortic coarctation.

Congestive cardiac failure occurs essentially in two groups of patients with aortic coarctation. In the first, failure occurs in early infancy. In these cases the question arises whether or not such patients should be operated upon early. Our belief at present is that operation should be postponed if the congestive failure responds well to digitalization. On the other hand, if the response to digitalization is not satisfactory, we believe that operation should be performed promptly in the infant group.

The vast majority of the patients who succumb to congestive

failure do so later in life, in the late 30's. Very often there are present other cardiac diseases which add to the factors causing congestive failure.

As far as rupture of the aorta is concerned, we should think of rupture of the aorta and rupture of a cerebral aneurysm very much in the same way. The anomaly is very much the same in both conditions.

If one reviews the data on rupture of the aorta, it is apparent that in the vast majority of cases (something like 80%) rupture occurs in the ascending aorta, not at the site of the coarctation. In approximately 20 per cent, rupture occurs just distal to the site of coarctation. It is extremely rare to see a ruptured aorta just proximal to the coarcted area.

There are many mechanical reasons for expecting rupture to occur just distal to a coarcted area but there are fewer reasons why this should occur in the ascending aorta. I hope Dr. Strauss will have something to say on this point. There are many who feel that there is a congenital abnormality in the wall of the aorta, very much the same as a congenital anomaly in the cerebral vessels which predisposes to the development of aneurysms. Hypertension also predisposes to some extent to vascular rupture. It should at once be added that there is no close correlation between the height of the blood pressure and the occurrence of rupture of the aorta. There is no significant difference in the blood pressure of patients who experience rupture of the aorta as compared with those who die of other causes. There is some difference among patients with rupture of a cerebral aneurysm but this difference may be interpreted in a different way. In most of these patients the elevated pressure is present after the rupture of the cerebral aneurysm and the possibility that the intracranial bleeding has caused the blood pressure rise cannot be excluded.

On this subject, I should like to mention in passing the question of pregnancy, which is of considerable interest and importance. There is one report which reviews the histories of 96 pregnant women with coarctation of the aorta. Eleven of these patients died of ruptured aorta during their pregnancy. In most of these cases, rupture occured just before or during labor, and there are many who feel that this anomaly should be corrected before pregnancy becomes far advanced.

In reviewing some of the data on intracranial lesions associated with coarctation of the aorta, we find that the average age of death is reported in two large series, as being 28 years: the range being

from a youngster of eleven years, on up to 54 years of age. In eleven cases which were reported, in which there was proof of rupture at autopsy, four of the patients had multiple aneurysms, varying in size from extremely small, to some that were the size of a walnut. There were four additional patients who had aneurysms which had not ruptured. One patient who had a previously-ruptured cerebral aneurysm which continued to leak, died of another cause. There were three patients who were believed to have died of a ruptured aneurysm, but in these cases no post mortem verification of this belief was found.

It is interesting to note that the aneurysms usually involve (or are near) a branch of the Circle of Willis at arterial junctions or bifurcations. In this relatively small group, all the ruptured aneurysms arose from intracranial branches of the internal carotid artery. Some of the unruptured aneurysms involved the basilar arteries or one of the vertebral arteries.

We hope that the incidence of aneurysmal rupture will decrease with early, elective correction of aortic coarctation. However, I think we all must anticipate that congenital aneurysms will be present in some of these patients, and this will not be influenced by correction of the coarctation. On the other hand, as Dr. Steinfeld has said, most of us feel that there must be some correlation with the presence of hypertension and actual aneurysmal rupture, although there is no exact correlation, the incidence of rupture should be decreased markedly by early correction of the coarctation.

On the question of management of such a patient as the one presented today, very little can be said with regard to the possibility of survival, because surgical repair of the coarctation in the face of bleeding from a ruptured intracranial aneurysm has very rarely been attempted.

My own feeling is that as soon as possible, one should attempt (once this accident has occurred) to keep blood pressure levels at a reasonable point. This was attempted in our patient with serpasil.

I think the second thing that should be considered is the question of use of hypothermia, in an attempt to do two things: to control blood pressure to some extent, and to control intracranial bleeding and any associated edema that may be present.

I think the coarctation should be operated upon "relatively early" in this situation (I don't know how early is early). I think that if the symptoms had subsided and one believed that the leak was under control, I should be inclined to do this within a week or two of rupture of the aneurysm.

If the patient continued to bleed, I think one's hand might be forced. I think that one would have to be extremely careful in the surgical management of the coarctation to do everything possible to keep the patient from developing extremely marked hypertension during the surgical procedure; we know that when the clamps are put on the aorta during the period of resection, there occurs very marked systemic hypertension. This may cause a critical situation at this time. One would have to be extremely careful to do everything possible to keep the patient as nearly normotensive as possible. Therefore, I am very much in favor of carrying out the repair of the coarctation under hypothermia. I would also use drugs such as serpasil.

On the other hand, one also runs the great risk—of which one must be aware—that these patients may become extremely hypotensive. One must walk this tightrope with great care.

I am not certain about the treatment of the bleeding aneurysm. I must say my own preference would be the correction of the hypertension first.

DR. HODES: Thank you very much, Dr. Blumenthal.

I should like to pass this x-ray film around because it does show notching of the ribs very clearly, and I don't think this has been referred to thus far in the discussion.

I should like to say that the remarks I shall make represent in part, my recollection of a rather detailed discussion about this subject which I had yesterday with the eminent neurologist, Dr. Sidney Carter of the Neurologic Institute of Columbia University.

First, with regard to hypothermia, Dr. Carter thinks this ought to be carried out at once. This agrees with Dr. Blumenthal's opinion, and with my own. The reason for instituting hypothermia at this moment, before anything else is done, is that there may be a favorable influence of hypothermia on the bleeding of the aneurysm, and there may be a minimizing of any brain injury due to the bleeding.

The next thing that Dr. Carter thinks ought to be done is to carry out the step which we've debated since this child was admitted. This is to perform an intra-carotid angiogram as soon as the child is put into a hypothermic state.

There is a difference of opinion regarding intracranial bleeding or intracranial aneurysms in general, in this wise: some advocate that an angiogram be done immediately and that operative steps be taken if the aneurysm is accessible to surgery. Others have preferred to wait for a period of a week or ten days in the hope that there might be some healing before an angiogram is done. Dr. Carter feels that

his experience indicates that everything is to be gained by doing the angiogram early, so that he would recommend that we carry out this procedure as soon as the child is hypothermic.

One might ask, "what are the reasons for doing the angiogram"? There are several good reasons. In the first place, one would like to know whether these are multiple aneurysms, as there frequently are in this situation. Knowledge on this point would help in determining the future course. A second important point is that the localization of the aneurysm is very important. We have no localization now, and we might also learn whether or not the aneurysm is accessible to surgical treatment. Dr. Carter thinks that there is considerable danger if surgery is undertaken without the angiogram. The neurosurgeon does not know where to look. Under these circumstances, there is a temptation to continue the search for the aneurysm beyond the safe point. It is important to have the scope of the possible operation outlined as far as it can be before the operation begins.

For example, the angiogram may show that there are multiple aneurysms, or that a single aneurysm is present, but that it is in a location which makes it inoperable. In either of these cases, we would abandon the idea of an intracranial operation, and proceed at once with the repair of the coarctation. And this brings us to the main question: whether the coarctation operation, or the intracranial operation should be done first—assuming that we have found an aneurysm which we think we can have corrected surgically.

Dr. Carter's opinion on this important point is the opposite of Dr. Blumenthal's, and this might be expected. One is a cardiologist and the other a neurologist. Dr. Carter feels quite firmly that the operation for the aneurysm, if the angiogram shows that it is single and is accessible, should be done first. He gives two reasons for this view. The first is that the immediate cause of death, if it occurs, will be further bleeding of this aneurysm, or another one, that the coarctation operation is not in itself essential, at least as far as the immediate saving of life is concerned. Dr. Carter thinks also that the coarctation operation would contribute to further bleeding of the aneurysm, and Dr. Blumenthal has himself explained how this might be brought about. It might be that during the course of the operation there will be a rise in blood pressure which may be fatal. Also Dr. Carter has the feeling, which I'm going to talk about in a minute, that the bleeding is not entirely mechanical. It is not due entirely, perhaps, to the effect upon abnormal intracranial vessels of the mechanical blockage of blood flow due to the construction in

the aorta. The hypertension may be due to reflex as well as mechanical factors. In other words, if one causes injury to the child in any way, emotional trauma for instance, there might be caused hypertension beyond that which is already present; this might cause further bleeding. Dr. Carter thinks that we should proceed in the order mentioned: hypothermia, angiogram, operation on an aneurysm if it is accessible, and then, after recovery from that operation, the coarctation operation should be undertaken.

I have said that there may be factors other than simple mechanical ones, and I think there may be some indication for this in the experience we had in this case with serpasil. There was a fall in blood pressure which we believe may be attributed perhaps to the effect of the serpasil; this may be taken as indicating that there are some reflex mechanisms at least partially involved here. This may be an error, but it seems to me at least to be a plausible theory. When thinking about aortic coarctation, one is tempted to consider it as a purely mechanical problem. From the discussion, we have learned that there are probably other factors involved. It has been mentioned already that renal problems may arise, and I was going to ask whether Dr. Blumenthal or Dr. Steinfeld would say something about the possible effects on renal blood flow of coarctation of the aorta. Perhaps there are available data on glomerular filtration rate in this condition.

DR. STEINFELD: A great deal depends on the type of coarctation which is present, as well as upon the age of the patient. In general, it is thought that the older patients do suffer some impairment of renal blood flow. Younger patients probably do not. There has been some experimental work to show that the kidneys are probably involved, but only in adults.

Scott and his co-workers carried out several experiments on dogs, in which one of the kidneys was grafted to a neck artery, leaving the other kidney in its normal position. Scott then produced coarctation of the aorta, thus subjecting the normally-situated kidney to a reduced blood flow. Hypertension occurred. Allowing the kidney connected with the neck vessel to remain intact, Scott next removed the normal kidney and the hypertension disappeared. This suggests that the hypertension produced by the coarctation may have been due to a "renal ischemia" mechanism.

DR. HODES: I should like to ask a question which is pertinent to this particular point. Does anybody know the mechanism of the fluctation in the blood pressure in children with this disease, because that bears upon the type of therapy that we might use. We think

that serpasil would be helpful, but we do not know what factors cause the hypertensive crises.

DR. BLUMENTHAL: I don't think anybody knows the mechanism, although the question has been discussed at great length. It should be noted that very commonly following resection of a coarctation, there are tremendous fluctations of blood pressures. It is not uncommon, as the chest is being closed, to have very marked and very severe hypotension develop suddenly. Many patients have required norepinephrine for a relatively long period. One can only theorize, to the best of my knowledge, as to the possible causes of these blood pressure variations.

DR. HODES: I should think it is possible that as a result of mechanical interference with the normal blood flow, there are reflex cerebral mechanisms at work here. This is what you imply by your citing of the hypotension which may occur immediately following operation.

DR. BLUMENTHAL: That is right. I think most people have the feeling that the causes of the hypertension in coarctation are probably multiple. There are probably reflex factors, and renal factors, as well as the factor of mechanical obstruction.

DR. HODES: Dr. Blumenthal, in view of our discussion, it seems to me that an intracranial attempt at controlling the situation is the proper procedure now. Have you changed your mind following the presentation of all the points of view which have been expressed?

DR. BLUMENTHAL: Yes, I would be very much influenced by what Dr. Carter had to say with regard to the possibility of achieving a good result.

DR. HODES: Do you think, for example, that if the angiogram shows a single aneurysm which is accessible, you would be willing to proceed with the intracranial operation first?

DR. BLUMENTHAL: That is right.

DR. HODES: Dr. Fischer, would you like to comment?

DR. FISCHER: Only to say that our neurologist, and our neurosurgeon have the same feeling as Dr. Carter. I should like to say also that much stress has been placed on the hypertension in this child as a possible cause of the rupture of the cerebral vessels. However, we all agree that there must be a congenital defect in the wall of the vessel, because hypertension occurs in many other conditions without causing vascular rupture.

DR. HODES: I think Dr. Fischer's point ought to be emphasized, that intracranial bleeding would not have happened if this child had normal blood vessels. We started out with Dr. Steinfeld's discus-

sion that this is a child with at least two anomalies that we are aware of. Dr. Gross?

DR. GROSS: If we were to do the coarctation operation first, would it is possible to do a bypass of the coarcted area, so that we would not increase the intracranial pressure?

DR. BLUMENTHAL: I think that would be a very inadequate surgical procedure, and I would almost prefer to do nothing unless the boy was really in extremis.

DR. HODES: Dr. Henley?

DR. HENLEY: Dr. Hodes, would you tell us something about the morbidity and mortality of operations on these aneurysms.

DR. HODES: I cannot quote exact figures; perhaps others can supply them, but I think the outcome depends upon the location of the aneurysm. Operation for cerebral aneurysm is still regarded as a serious operation. However, in a case like this, we must operate. Results are now much better than those which follow no intervention. With the angiogram, we have a good chance of coming to a decision which may be of use to the patient.

So, intracranial bleeding is not considered a hopeless kind of thing. It should be pinpointed and located as accurately as we can do it, and proper operative procedures should be carried out.

DR. GRIBETZ: I want to emphasize the point that hypertension must have something to do with this condition as well as the anomaly of the vessel. Could we ask, Dr. Blumenthal, whether the results in spontaneous sub-arachnoid hemorrhage in young adolescents who don't have pre-existing hypertension are somewhat better in terms of recovery in children?

DR. BLUMENTHAL: I don't think I can answer that question.

DR. FISCHER: Are you doing the angiogram today?

DR. HODES: Yes. The child will be made hypothermic, and we shall do the angiogram today.

VOICE: Must we expose the child to the angiogram now?

DR. HODES: I think we must, because the risk is much greater if we don't do it. I think we need an angiogram after hypothermia, and operation, if the aneurysm is accessible to the operation.

VOICE: Does hypothermia always make it possible to proceed with surgery?

DR. HODES: No, I don't think it always does, but I think the risk to the patient is reduced and this is coming to be recognized as a regular method of treatment in these cases.

VOICE: If the aneurysm is accessible, exactly what is the surgical technique?

DR. HODES: The only one I'm familiar with is closing it with a silver metal clip of some kind. Recently, a method of coating the aneurysm with a plastic has been developed. In some cases, at least, this prevents further bleeding for a considerable period. We don't have a neurosurgeon here at the conference, unfortunately.

DR. BLUMENTHAL: I should like to emphasize the importance of hypothermia in this situation. I think we've seen some good results in this situation as part of good over-all management of the patient.

VOICE: Dr. Hodes, is there some defect of the wall of the aorta, just distal to the coarctation, in this case?

DR. HODES: Dr. Steinfeld, can you answer this question?

DR. STEINFELD: Many of the individuals who have coarctation have a hypoplastic aorta. They may have a poorly-developed aortic media, in the ascending aorta, in the part of the aorta which is distal to the coarctation. One must consider the presence of a "jet" as a possible cause for this.

The coarctation itself might be described as a semi-lunar infolding of the wall of the aorta. As a result, there may be an eccentric narrow lumen, and secondary to this a jet will be formed, which will hit the aortic wall on one side. In this area there is a site of marked stress to the aortic wall and an aneurysm may form quite easily.

DR. BLUMENTHAL: This is really the same as post-traumatic dilatation that he finds in pulmonary stenosis. There have been some interesting experiments performed whereby one puts a tie through an ordinary rubber tube, creating a marked obstruction with continuing flow. The tube will give way just distal to the tie. I think the problem in that situation is mechanical and is different from a congenital defect.

DR. HODES: Dr. Blumenthal, do you or Dr. Steinfeld have some current statistics about the success of operations for coarctation similar to that of our case, leaving the intracranial problem out of the question for the moment?

DR. BLUMENTHAL: The mortality rate has been dropping over the past decade and it has been interesting to see the difference between the results in the first patients reported by Dr. Gross, and in his last series of patients.

At the present time, in good surgical hands the mortality rate in operations for uncomplicated coarctation of the aorta is less than 5%.

DR. HODES: If there aren't any other questions, I think our time is up. I should like to thank Dr. Blumenthal, Dr. Steinfeld, and Dr. Warren for the discussion.

Current Literature

Edited by

MICHAEL A. BRESCIA, M.D.

PHYSICIAN'S HANDBOOK, 10TH REVISED EDITION, by Marcus A. Krupp, M.D., Norman J. Sweet, M.D., Ernest Jawetz, M.D., and Charles D. Armstrong, M.D. Pages 488, Lange Medical Publications, Los Altos, California, 1958. Price $3.00.

The purpose of this handbook has been to summarize tersely, clearly and comprehensively, diagnostic procedures and factual data which a physician must have available. At the same time, the scope of this manual has been extended so as to be a serviceable pocket reference for many types of medical practice.

This handbook includes a relatively complete laboratory manual, the common clinical tests, and such other factual information as is more readily forgotten. Emphasis is placed on clinical factors, such as, the significance of abnormal laboratory test findings. This will be noted in sections on the urine, blood and liver tests, the hormones; and diagnosis of poisonings. A separate chapter discusses the biologic products for prophylaxis and therapy and alcoholic intoxication. Many of the new drugs are discussed including the indications, toxicity and treatment with dosages.

The Physician's Handbook serves to provide the student and practicing physician with a ready reference of practical value in his daily hospital program and medical practice. I recommend this manual to all students, internes, residents and physicians regardless of specialty. JOSEPH M. COVELLI, M.D.

PEDIATRICS FOR NURSES, ARTHUR G. WATKINS. The Williams & Wilkins Co. Baltimore, Md.—Exclusive U.S. Agents. Cloth pp. 220 Pictorial Illustrations. Price: $3.75.

Within the last ten years, the changing trends in pediatrics have been so remarkable that many concepts of Pediatrics for Nurses have been completely revised. We have new diseases and new syndromes. With the recent attention given to rehabilitation, social, economic and environmental factors in disease; we find preventive medicine broadening the scope of treatment in the hospital wards. Since the causes for illness begin with intrauterine life—to birth and carry on to independence, continuous public health teaching is necessary in prenatal care to avoid pediatric disorders.

More recently consideration is concentrated on relationships between the parent and child and again on sibling adjustment.

Children with diseases such as pyloric stenosis and inguinal hernias are now hospitalized for 48 hours with the surgical follow up care rendered at home. The home therapy prevents the hazard of cross infection, anxiety and stress. This type of advanced surgical postoperative treatment hasn't been completely accepted in America as well as it has been in Wales.

The text has an excellent index and may be of practical service to student nurses and graduated registered nurses. It is entirely clinical in its approach. The treatment of diseases are touched on briefly. Techniques in nursing care and communicable diseases are omitted.

This book has valuable information as a guide to any nurse who desires greater comprehension of pediatric diseases and disorders. It is simply written, the disorders are separated into the nine functional systems of anatomy and physiology. I recommend it as an additional text for students in training and hospital libraries or public health child health centers. ELSIE MECHTA, R.N.

MANUAL OF PEDIATRIC PHYSICAL DIAGNOSIS by Lewis A. Barness, M.D., pages 179, cardboard cover, The Year-Book Publishers Inc., Chicago, Ill., 1957, Price $4.00.

This manual originated as a series of mimeographed notes to second year students on pediatric physical examination and diagnosis. These notes were expanded and compiled into a text which is clear and concise not only to the medical student in his study of pediatrics, but helpful to the general practitioner and pediatrician in his every day examination of infants and children. The reading of this manual is not only refreshing to the physician, but many hints and signs are presented which I am sure, many of us have forgotten.

This is a manual of the special methods used in pediatric physical diagnosis. Examples of disease states are given to present a better understanding of the sign under discussion. The author emphasizes the fact that there is no "routine" physical examination of a child. Each examination is individualized. The many physical differences and variations among a group of children, makes the examiner more alert to the broad spectrum in the term "normal".

In this manual of pediatric physical diagnosis will be found the usual observations noted with inspection, percussion, palpation and

auscultation. Discussion is according to the systems of the body and anatomical orientation, such as, head, neck, chest, heart, abdomen, extremities, neurological etc. Examination of the newborn, the premature and the child are discussed separately.

This small text book is definitely valuable not only to students but to all physicians in general practice and in pediatrics.

JOSEPH M. COVELLI, M.D.

NERVOESE VERHALTUNHSSTOERUNGEN BEIM SCHULKIND; EINE MEDIZINISCH-SOZIOLOGISCHE UNTERSUCHUNG, by Gustav-Adolf Von Hœrnack. Paper Pg. 93. Price $2.35. Stuttgart, Germany. George Thieme, 1958.

The author attempts to correlate behavior disorders of the 11 to 12-year-old child with environmental factors such as broken homes, divorce, part-time work of mothers etc. The book represents a follow-up of studies started several years ago of children entering elementary school. Data were obtained from teachers, social workers, mothers and school physicians. The percentage of behavior disorders was much higher in the poorer classes of the population compared with the socially better situated ones. The influence of illegitimacy, broken homes was pronounced especially in cases of sleep disturbances, hyperkineses, enureses and general motor restlessness. These disorders were also more common among children of "nervoue" mothers and a viscious cycle was thus established.

Children raised in suburban homes showed a small but statistically significant lower rate of occurrence of so-called vegetative neurosis such as recurrent headaches, fainting spells etc. compared to those brought up in a large city. Motor disturbances such as tics, stuttering, gnawing of teeth were less dependent on environmental factors. Acts of delinquency such as running away from home and school, stealing, brutality often have a genetic background, occasionally they are due to neurosis. Their rate of occurrence in broken homes and incomplete families was found to be 12 times higher as compared to children from good families. A number of behavior disorders showed a definite increase following start of school.

The author also discusses the relationship between school marks, social background and environmental factors of these children. The highest percentage of children with lack of concentration and poor school marks was found among children of "nervous" mothers. Lack of concentration and hyperkinesis are central problems of school children.

M. WEICHSEL, M.D.

HENDREN, W. H. III, HAGGERTY, R. J.: STAPHYLOCOCCIC PNEUMONIA IN INFANCY AND CHILDHOOD. ANALYSIS OF SEVENTY-FIVE CASES. (Journal American Medical Association 168:6 Sept. 6, 1958).

Seventy-five consecutive patients, recently observed with primary staphylococcic pneumonia were treated by the medical and surgical services of the Children's Medical Center. Bacteriological cultures of hemolytic Staphyloccoccus pyogenes var. aureus were obtained from empyema fluid or the lung in 40 patients (53%) in this series. Typical x-ray findings of staphylococcic pneumonia were present in all other patients. Distinctive roentgenographic features included the formation of usually multiple, small, pulmonary, radiolucent areas, pneumatocele formation, empyema, pneumothorax, and rapid change from one appearance to another.

Males outnumbered females by a 2:1 ratio. Although principally a disease of infants, occasionally cases occurred in older children. A significant number of patients had concomitant cutaneous staphylococcic infections. Many had previously received antibiotic treatment. Physical signs of pneumonia were not present initially in some patients.

We stress the need, in treatment of these patients, for vigorous supportive care, blood transfusions, prolonged treatment with specific antibiotics, and, frequently, closed-tube thoracotomy. Closed-tube thoracotomy must be regarded as a conservative measure which is simple to perform and often lifesaving. Among the indications for insertion of a closed-thoracotomy tube are a significant collection of pleural fluid and tension pneumothorax. Attention should be paid to the proper technique of such drainage.

Enzymes have been employed in some patients whose illness is complicated by empyema. Differential diagnostic considerations which have been encountered include abdominal emergencies, diaphragmatic hernia, and intrathoracic tumor. Nine deaths occurred in this series. Staphylococcic pneumonia is an important entity with which physician and surgeon alike should be familiar, since a high proportion of patients require the services of both.

get them out of bed quickly...safely

with tri-sulfanyl

sulfonamide therapy at its best

rapid, maximum recovery assured...
because of rapid, prolonged high blood and tissue levels of triple sulfa mixtures.

worry-free therapy... high urine solubility makes risk of crystalluria virtually negligible. As specific as antibiotics in many infections, but avoids certain of their complications. Danger of moniliasis, gastric upsets, bacterial resistance, sensitivity, blood dyscrasia, etc. reduced to a minimum.

the candy-like flavor of Tri-Sulfanyl syrup appeals to all.

Each 5 cc. of Tri-Sulfanyl syrup, or each tablet contains 0.5 Gm. of total sulfas (equal parts of sulfadiazine, sulfamerazine and sulfathiazole) with 0.375 Gm. of sodium citrate (in syrup only).

4 oz., 16 oz. and gallon syrup; 100 and 500 tablets.

SAMPLES and new literature on request.

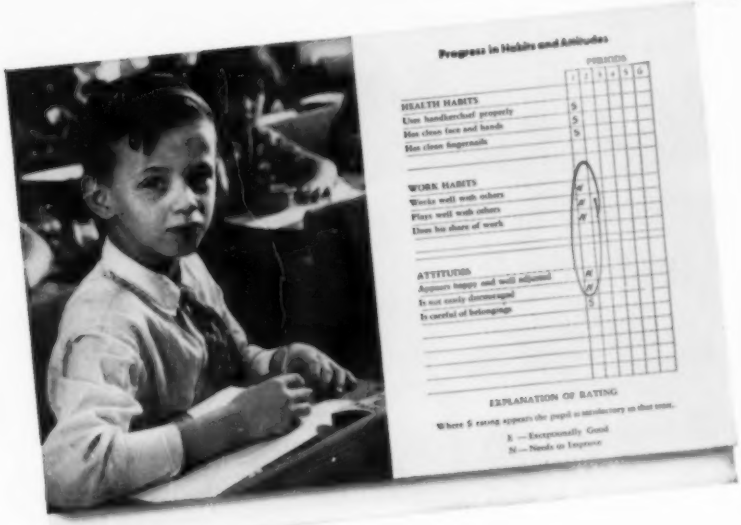
arlington-funk laboratories
division of U. S. Vitamin Corporation, 250 East 43rd St., New York 17, N.Y.

THE TRANQUILIZER UNIQUE IN PEDIATRICS

REPORT CARD DIAGNOSIS: ANXIETY

PRESCRIPTION: ATARAX®

(brand of hydroxyzine)



FOR SMALL PATIENTS WITH LARGE PROBLEMS **temper tantrums–nervous tics–vomiting–nervousness–stuttering–asthma–hyperkinesia–nightmares–enuresis**

These manifestations of emotional disturbances respond to the remarkable calming effect of ATARAX.[1,2] Bayart also reports that children receiving ATARAX obtained "distinctly more regular and more favorable scholastic results" through freedom from anxiety and tension.[2]

Safe, even in high doses

Ayd recommends ATARAX particularly for school children, since many "have tolerated well doses in excess of the average therapeutic dose."[3]

Dosage: Children (3 to 6 years), usually one teaspoon syrup or one 10 mg. tablet t.i.d.; (over 6 years), usually two teaspoons syrup or two 10 mg. tablets t.i.d.

Supplied: Tablets, tiny 10 mg. (orange), 25 mg. (green), and 100 mg. (red), bottles of 100. Syrup, 10 mg. per tsp., pint bottles. Prescription only.

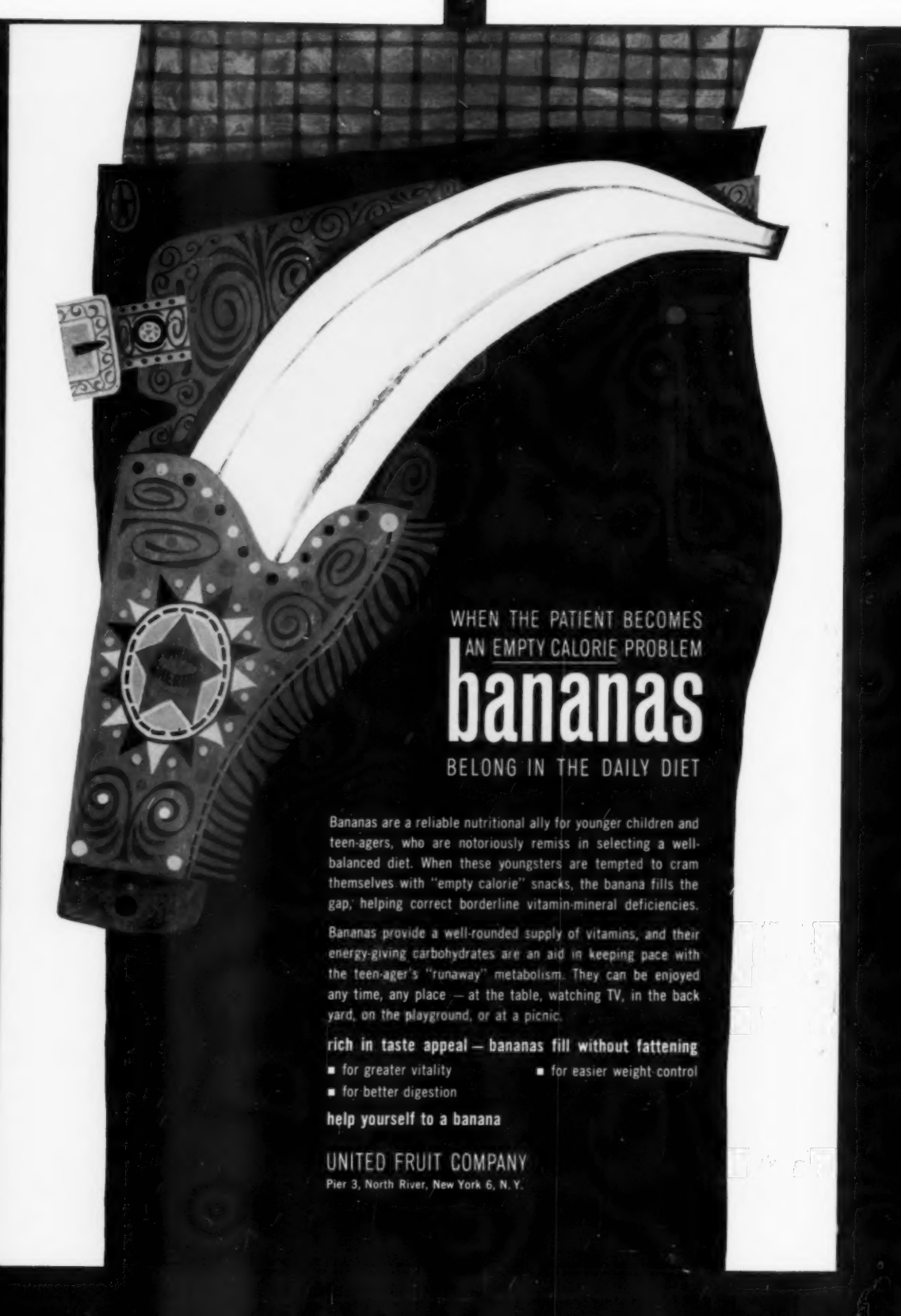
References: 1. Nathan, L. A., and Andelman, B. M.: Illinois M. J. *112*:171 (Oct.) 1957. 2. Bayart, J.: Presented at International Congress of Pediatrics, Copenhagen, Denmark, July 22-27, 1956. 3. Ayd, F. J., Jr.: California Med. *87*:75 (Aug.) 1957.

℞ **good-tasting ATARAX Syrup** makes administration easy for the parent — pleasant for the child.

ROERIG ℞

New York 17, N. Y.
Division, Chas. Pfizer & Co., Inc.
Science for the World's Well-Being

ATARAX®


WHEN THE PATIENT BECOMES
AN EMPTY CALORIE PROBLEM
bananas
BELONG IN THE DAILY DIET
Bananas are a reliable nutritional ally for younger children and teen-agers, who are notoriously remiss in selecting a well-balanced diet. When these youngsters are tempted to cram themselves with "empty calorie" snacks, the banana fills the gap, helping correct borderline vitamin-mineral deficiencies.
Bananas provide a well-rounded supply of vitamins, and their energy-giving carbohydrates are an aid in keeping pace with the teen-ager's "runaway" metabolism. They can be enjoyed any time, any place — at the table, watching TV, in the back yard, on the playground, or at a picnic.
rich in taste appeal — bananas fill without fattening
■ for greater vitality
■ for better digestion
■ for easier weight control
help yourself to a banana
UNITED FRUIT COMPANY
Pier 3, North River, New York 6, N. Y.